New Orleans Medical
and
Surgical Journal

$3.00 Per Annum, 35c Per Copy
Vol. 101—No. 5

NOVEMBER, 1948

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

ON THE ETIOLOGY OF NEPHRITIS*

J. M. HAYMAN, JR., M. D.†

CLEVELAND, OHIO

In order to discuss the etiology of nephritis we must define what kind of kidney disease we are talking about. This requires some statement of the classification of renal disease which is far from satisfactory or settled. The general grouping proposed by Volhard and Fahr,[1] and by Addis[2] in slightly different terms, is at present pretty generally accepted. This includes three great groups—inflammatory, vascular, and degenerative or nephrotic—the first two being primarily disease of the glomeruli, the third manifested chiefly by changes in the renal tubules. Several thoughtful modifications or elaborations have been made in the light of further study, notably by Bell[3] and very recently by Leiter.[4] (Table I) Both of these remove the so-called lipoid nephrosis from the group of tubular diseases, since abnormal glomerular permeability to plasma proteins is probably the primary defect in the disease. Bell calls it membranous nephritis, and Leiter, glomerulonephrosis. Either grouping must be regarded as tentative until it is determined whether lipoid nephrosis is a disease entity or simply a peculiar stage in the course of chronic glomerulonephritis. Certainly adults with the nephrotic syndrome usually lose their edema, develop hypertension and renal insufficiency, and die as chronic nephritics. In children many more apparently recover completely—the others used to die of intercurrent infection, especially pneumococcic, but now with antibiotics

TABLE I

CLASSIFICATIONS OF RENAL DISEASE

BELL	LEITER
1. Glomerular Diseases	1. Glomerulonephritis (Acute; chronic)
a) Proliferative glomerulonephritis (Acute; chronic)	2. Glomerulonephrosis (Lipoid; amyloid)
b) Membranous glomerulonephritis (Lipoid nephrosis)	3. Glomerulosclerosis (Arteriolar; diabetic)
c) Amyloid disease	4. Glomerulitis (Toxic; embolic; allergic)
d) Toxemias of pregnancy	5. Pyelonephritis (Acute; chronic)
2. Tubular Diseases	6. Vascular (Arteriosclerotic; allergic)
a) Degenerative	7. Tubular (Necrotizing: obstructive; degenerative)
b) Obstructive	
3. Diseases of Interstitial Tissue	
a) Cortical abscess	
b) Pyelonephritis	
4. Diseases of Vascular System	

*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 13, 1948.

†From the Department of Medicine, Western Reserve University, Cleveland, Ohio.

more go on to develop chronic nephritis. It is the pediatricians in particular who hold out for lipoid nephrosis as a metabolic disease, but even in this group opinion is far from unanimous.

Ellis[5] believes that there are two types of inflammatory Bright's disease. One type develops acutely after a recognized upper respiratory tract infection, and is most common in the first two decades. Fever, malaise, headache, and such general symptoms are common. Hematuria is constant, but edema is of short duration. The prognosis is good, recovery occurring in some 80 per cent. The other type is characterized by insidious onset, without recognized preceding infection, edema is persistent, and prognosis poor. There is, however, considerable overlapping, and neither the course nor pathologic picture permits determination in which group many cases should be placed. This division has not been widely accepted in this country. It seems that the two types may simply represent extreme variations which the same disease may show.

The foundation of any attempt to interpret the mechanism by which acute glomerulonephritis develops must be based on several well known characteristics of the disease. (1) Its onset is almost always preceded by infection with hemolytic streptococci. (2) Only a small proportion of patients with such infections develop frank nephritis. Some abnormalities of the urine, as measured by Addis counts, are present in a much higher proportion. (3) A latent period of 12-25 days intervenes between the onset of the infection and the onset of the nephritis. (4) This latent period is much shorter in exacerbations of a previously existing nephritis. (5) Bacteria can rarely be found in urine or kidneys of patients with glomerulonephritis.

These facts point strongly to the view that acute nephritis in man represents an allergic reaction occurring in the kidneys, presumably as the result of an auto-immune response to kidney protein rendered antigenic by the toxic or denaturant action of some streptococcal product or combined streptococcal-tissue product.

Experimentally renal lesions have been produced by injection of nephrotoxic sera. In 1934 Masugi[6] obtained lesions closely simulating human nephritis by injecting rabbits with antikidney serum prepared in the duck. However, Kay[7] has pointed out that even the Masugi nephritis cannot be a simple kidney antigen-kidney antibody reaction, because there is a latent period before the appearance of a renal lesion. It is impossible to produce antikidney antibodies in the same species by the Masugi technic. A further step was made by the Caveltis[8] who suggested that the kidney acts as a hapten and the streptococcus as the protein carrier in an auto-antibody response. They produced lesions in rats which closely simulated both acute and chronic nephritis by injection of mixtures of killed streptococci and kidney extracts.

The human situation may be much more complex than such experiments would indicate. Kay, Luchesi and Rutherford[9] were unable to demonstrate antikidney antibodies in the serum of patients with acute nephritis by complement fixation or precipitins. There is much evidence that in acute nephritis there is disease of small vessels throughout the body, and not only in the kidney. The frequency of cardiac dilatation, unrelated to hypertension, electrocardiographic changes, and the perivascular lesions and acute arteriolitis reported by Stone[10] and by Feller and Hurevitz[11] in biopsies of muscle support this belief. This does not exclude the whole hypothesis. A combined or complex autoantigen may be responsible for the diffuse lesions similar to the polyarteritis and periarteritis-nodosa like lesions produced by Rich[12] in rabbits by a sulfa drug and horse serum.

One point in the experiments of the Caveltis' is of particular importance. They produced chronic nephritis in rats by a single injection of streptococci and kidney extract. Some animals died in the acute phase, some apparently recovered but some after subsidence of acute symptoms

developed a chronic nephritis with decrease in function. That is, a chronic nephritis followed a single insult, without repeated introduction of antigen. This to me fits in with human disease, where the chronic nephritis progresses without any discoverable infection being present.

That the kidneys are somehow involved in essential hypertension and hypertensive vascular disease is pretty generally accepted. All the evidence indicates that arteriolar disease is more common and more severe in the kidneys of hypertensives than in normotensives, and in the hypertensives it is more severe in the kidneys than in other organs (except spleen). The question, however, is whether this arteriolar nephrosclerosis is the primary factor in "essential" hypertension, or is simply part of the functional vascular derangement produced by persistent hypertension.

Goldblatt[14] believes that the arteriosclerosis is primary, leads to a disturbance in renal hemodynamics, this in turn to an increased formation of renin, which after activation, leads to generalized vasoconstriction and hence to hypertension. Others, as Goldring and Chasis,[15] Katz and Leiter,[16] and Page and Corcoran[17] believe that the initiating factor of the general vasoconstriction is unknown, perhaps psychogenic or neurogenic, and that the disturbance in renal hemodynamics is part of this functional vascular derangement. In favor of this view they point to the symmetrical changes in glomerular and tubular function as measured by clearance methods, the bilaterality of pathologic changes, and the early lability of the blood pressure. On the other hand, there are many similarities between the experimental and clinical disease. In both the increased tension is the result of a generalized increase of peripheral resistance. In both there may be no detectable change in renal function, or severe renal insufficiency, depending in the dog on the degree of constriction of renal arteries and in man on the amount of renal tissue remaining. In both, cardiac rate, output, pulmonary artery pressure, and venous pressure are normal. In both, hypertension associated with unilateral disease may be cured by removal of the diseased kidney provided the other is normal.

Both theories have a serious gap. The Goldblatt hypothesis does not explain why the renal arterioles get sclerotic in the first place and the primary neurogenic hypothesis does not explain the much g r e a t e r change in renal than in other vessels. The objection of Goldring and Chasis that the normal clearance and estimated renal blood flow exclude "renal ischemia" is not valid, for many of Goldblatt's dogs had normal renal function. Possibly the elevated blood pressure maintains a normal renal flow in spite of mechanical obstruction. Certainly the lability of the blood pressure in early stages suggests a nervous or nervous-hormonal mechanism. Much attention is again being given to the role of salt, or rather sodium, retention in the production of both experimental and human hypertension.[18] What this means in regard to the relation of adrenal cortical hormones to hypertension cannot be clearly seen at present. Perera[19] and his associates have shown recently that patients with essential hypertension respond to sodium restriction as though they had excessive desoxycorticosterone in them. This is certainly a fertile field for those who believe in the psychogenic or neuro-endocrine theory of the origin of essential hypertension.[20]

That heavy metals and certain other poisons produce lesions in fairly specific parts of the tubules is well known. Recently much attention has been directed to a similar clinical picture occurring in a wide variety of conditions—hemorrhage, surgical shock, crushing injuries, various infections, etc. Some of these show the most striking degeneration in the distal tubule, rather than in the proximal as with heavy metals, and for these Lucke[21] has used the term "lower nephron nephrosis" and Malgraith[22] "tubovascular syndrome." The dividing line between these conditions and the organic chemical nephroses is in many ways a tenuous one. The important unifying feature is what McCance and Lawrence[23] have aptly called "functional disor-

ganization" of the kidney. The renal tubule loses its highly selective ability to reabsorb certain elements and to discard others in the glomerular filtrate. Salt, nonprotein nitrogen, glucose, water, acids, bases, etc., may diffuse back completely through the disorganized tubule. The result is the passage of only small volumes of dilute urine, or anuria, and the rapid development of uremia superimposed on the patients' other troubles.

Aside from the heavy metal poisons, the mechanism producing these nephroses is apparently too long a period of renal anoxia, resulting from low blood pressure and diminished renal flow.[24] Trueta[25] on the basis of extensive experiments on rabbits believes that in shock, etc., blood is directed from its normal cortical course to those glomeruli lying nearer the medulla, with a resulting cortical ischemia. This has been called a renal shunt but it is not a shunt in the sense of opening up of channels not usually perfused but rather the continuance of blood flow in only one of two channels which are both normally open. That an abnormally large volume of blood goes through the open channel is unproved. While Trueta did not measure blood flow, all his data indicate that it was reduced whenever cortical ischemia was present.

Early recognition and prompt treatment are essential, for there are only two alternatives in this condition; complete recovery or death from uremia. Chronic intermediate stages do not seem to occur. While the human kidney can stand even complete interruption of its arterial supply for twenty or thirty minutes, when cortical ischemia persists for twelve or twenty-four hours, severe functional if not histologic changes are produced in tubule cells.

There are other types of renal disease, with probably distinctive etiologies, for which the mechanisms are more or less understood. These include the intercapillary glomerulosclerosis of diabetes mellitus, the renal defect in Addison's disease, diabetic insipidus, rickets, Cushing's syndrome, and pyelonephritis, but a discussion of these is beyond the scope of this presentation.

SUMMARY

Recent studies have led to modifications of the classification of renal disease, permitting the recognition of noninflammatory or vascular glomerular lesions, the elimination of focal glomerulonephritis, and a better understanding of the diseases affecting the renal tubules primarily.

Glomerulonephritis is believed to be due to an allergic reaction manifested chiefly in the kidneys, and resulting from an abnormal response to infection, which is usually streptococcal. The importance of renal arteriolar sclerosis in essential hypertension is recognized, but whether this is primary, or the result of psychogenic or hormonal influences is unsettled. Aside from poisoning by heavy metals and organic compounds, the most important cause of tubular degeneration appears to be anoxia from diminished renal blood flow.

BIBLIOGRAPHY

1. Volhard, F., and Fahr, T.: Die Brightsche Nieren-Krankheit, Julius Springer Berlin, 1914.

2. Addis, T. and Oliver, J.: The renal lesion in Bright's disease, Lea and Febiger, Philadelphia, 1930.

3. Bell, E. T.: Renal diseases, Lea and Febiger, Philadelphia, 1946.

4. Leiter, L.: Renal diseases: some facts and problems, Ann. Int. Med., 28:229, 1948.

5. Ellis, A.: Natural history of Bright's disease, Lancet, 1:1, 1942.

6. Masugi, M.: Über die experimentelle glomerulonephritis durch das spezifische antimerenserum, Beitr. z. path. Anat. u. z. allg. Path., 92:429, 1934.

7. Kay, C. F.: The mechanism of a form of glomerulonephritis; nephrotoxic nephritis in rabbits, Am. J. Med. Sci., 204:483, 1942.

8. Cavelti, P. A., and Cavelti, E. S.: Studies on the pathogenesis of glomerular nephritis, Arch. Path., 39:148, 1945.

9. Kay, C. F., Luchesi, P. F. and Rutherford, R. B.: An experimental investigation of an immunologic mechanism as the cause of glomerulonephritis, J. Immunology, 42:369, 1943.

10. Stone, W. J.: Bright's disease and arterial hypertension, W. B. Saunders, Philadelphia, 1936.

11. Feller, A. E., and Hurevitz, H. M.: Am. Heart J., 16:568, 1938.

12. Rich, A. R.: Role of hypersensitivity in periarteritis nodosa, Bull. Johns Hopkins Hosp., 71:123, 1942.

13. Moritz, A. R., and Oldt, M. R.: Arteriolar sclerosis in hypertensive and nonhypertensive individuals, Am. J. Path., 13:679, 1937.

14. Goldblatt, H.: The renal origin of hypertension, Physiol. Rev., 27:120, 1947.

15. Goldring, W., and Chassis, H.: Hypertension and hypertensive disease, The Commonwealth Fund, New York, 1944.

16. Katz, L. N., and Leiter, L.: The present conception of essential hypertension, Psychosom. Med., 1:101, 1939.

17. Page, I. H., and Corcoran, A. C.: Arterial hyper-

tension; its diagnosis and treatment, Yr. Book Pub., Chicago, 1945.

18. Grollman, A., Harrison, R. T., Mason, M. F., Baxter, J., Compton, J., and Reichsman, F.: Sodium restriction in the diet for hypertension, J. A. M. A., 129:533, 1945.

19. Perera, G. A., and Blood, D. W.: The relationship of sodium chloride to hypertension, J. Clin. Invest., 26:1109, 1947.

20. Selye, H.: General adaptatio nsyndrome and diseases of adaptation, J. Clin. Endocrinol., 6:117, 1946.

21. Lucke, B.: Lower nephron nephrosis, Mil. Surgeon, 99:371, 1946.

22. Malgraith, B. G., Harvard, R. E., and Parsons, D. S.: Renal syndrome of wide distribution induced possibly by renal anoxia, Lancet, 2:293, 1945.

23. McCance, R. A., and Lawrence, R. D.: The secretion of urine in diabetic coma, Quart. J. Med., 4:53, 1935.

24. Selkurt, E. E.: Comparison of renal clearances with direct blood flow under control conditions and following renal ischemia, Am. J. Physiol., 145:376, 1946.

25. Trueta, J., Barclay, A. E., Franklin, K. J., Daniel, P. M., and Prichard, M. M. L.: Studies of the renal circulation, Charles C. Thomas, Springfield, 1947.

THE PATHOLOGICAL PHYSIOLOGY OF CHRONIC NEPHRITIS*

J. M. HAYMAN, JR., M. D.†
CLEVELAND, OHIO

Before discussing the physiology of chronic nephritis, it is necessary to recall a few anatomical facts. Each human kidney is made up of approximately a million and a quarter units or nephrons, each consisting of a glomerulus and its attached tubule. This number is present at birth, and in health persists throughout life. There is no regeneration or new formation of glomeruli. Once a glomerulus is destroyed by disease, the total glomerular equipment of the subject is reduced. The condition is quite different, however, with the epithelium of the convoluted tubules. Here regeneration takes place readily and rapidly, presumably from those cells which have escaped destruction.

In certain types of chronic nephritis (chronic glomerular nephritis and vascular disease or arteriolar nephrosclerosis) the kidneys are reduced in size, and examination of microscopic sections shows many destroyed glomeruli and hyaline scars. Neither the size of the kidney nor the number of visible scars bears any satisfactory relation to the duration of disease or the degree of impairment of kidney function. While many of the remaining glomeruli are seen to be more or less damaged, the tubular epithelium is frequently not strikingly abnormal. If the remaining nephrons functioned in the normal manner, such kidneys might be expected to excrete a small amount of normally concentrated urine. In fact, however, the urine volume is increased, and concentrating power is diminished or lost. Must it be assumed, therefore, that in addition to the apparent loss of a number of nephrons, those which remain are qualitatively abnormal?

One of the first questions to be answered in the study of the subject was whether the reduction in the size of the kidney in chronic Bright's disease was a measure of the reduction in the number of nephrons. This involved counting the number of glomeruli. Fortunately, Kunkel[1] had developed a method which with slight modification was suitable.

In 14 normal kidneys from patients aged one hour to 88 years, the total number of glomerular structures varied from 940,000 to 1,542,000. The mean was 1,282.8 $\pm$ 37.7 thousand.[2] Some few hyaline scars of destroyed glomeruli were identified in 10 of the 14 kidneys, particularly in the older subjects. In chronic glomerular nephritis and in vascular disease, however, the number of possibly patent glomeruli is usually reduced to below 500,000 and may be below 200,000 while the total number of recognizable glomerular structures, including scars, is likewise reduced. The reduction of the number of glomeruli was not paralleled by a corresponding reduction from the expected normal weight of the kidney. Nor could the reduction in the number of glomeruli be judged by the relative number of hyaline scars, for in some kidneys with a low total number of recognizable glomerular structures, the percentage of scars was relatively low.

The reduction in the total number of recognizable glomerular structures indicates that in chronic kidney disease many of these may disappear without leaving

*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 13, 1948.

†From the Department of Medicine, Western Reserve University, Cleveland, Ohio.

recognizable trace. The same result was obtained in rabbits whose kidneys had been injured by x-rays or by kinking of the ureter for a few days.

If a large proportion of the glomeruli in chronic renal disease can disappear without leaving a trace, the final histologic examination may give less information concerning the pathogenesis and severity of the disease than is commonly thought. It is not fair to assume that the changes affecting the remaining glomeruli are necessarily the same as those that occurred in the glomeruli that have disappeared.

Since it had been found that there was no correlation between kidney function during life and kidney weight after death, the next question was whether a relation did exist between kidney function and the number of remaining patent nephrons. Two commonly used tests of renal function, the urea clearance test of Möller, McIntosh and Van Slyke, and the maximum specific gravity or concentration test, were carefully carried out on a number of patients suffering from renal disease but without evidence of heart failure, and then the number of glomeruli was estimated at autopsy from a few days to several months later by the method just described.[3]

If the mean urea clearance, in percentage of normal, was plotted against the number of glomeruli per kidney, a reduction in clearance with a decreasing number of nephrons was apparent. The relation, however, was not a direct one, function being reduced more rapidly than the number of nephrons. In spite of considerable scattering, the points fell fairly well along an exponential curve concave upward. In contrast to this, when the renal mass had been reduced by subtotal nephrectomy in dogs, the percentage reduction in clearance was less than the reduction in glomeruli, so that the curve was convex upward.[4] In the dogs the remaining glomeruli were normal, while in the patients many at least of the remaining nephrons were more or less damaged. The pathologic changes may include not only a reduction in the area of filtering surface, but also a decrease in the permeability of the glomerular membrane, so that there is less filtrate for a given filtering area and capillary pressure.

The concentration test is a sensitive, reliable and easily performed test of renal function; but it is well known that the specific gravity of the urine may reach a minimum value of about 1.010 while the patient is still free from symptoms, and that with further progress of the disease, no further reduction in specific gravity occurs. Alving and Van Slyke[5] compared the urea clearance and concentration tests and found that both were equally good until the specific gravity of the urine had fallen to 1.010, which occurred when the value for the urea clearance had reached about 35 per cent of average normal. After this the clearance test alone showed the progress of the disease. These facts are confirmed by a correlation of the maximum specific gravity and the number of glomeruli. The shape of the curve is different from that afforded by a correlation of the urea clearance value and the number of glomeruli. It is approximately a horizontal line up to between 700,000 and 800,000 glomeruli per kidney, and then it rises at an angle of about 45° to the normal range. The point of apparent discontinuity, about 700,000 or 800,000 glomeruli per kidney, corresponds to about 35 per cent of normal urea clearance. The reason for this apparent discontinuity invites speculation. It may correspond to the point at which all the remaining glomeruli become continuously active.

There is little correlation between the systolic blood pressure and the estimated number of glomeruli, except that no patient was encountered with less than 700,000 glomeruli per kidney who had a systolic pressure below 150 millimeters of mercury. On the other hand, a person with a normal number of injectable glomeruli may have an elevated blood pressure. The data on hand, however, at least suggest that when the glomeruli have been reduced to 700,000 to 800,000 per kidney, there is some change associated with the presence of elevated blood pressure and loss of concentrating power. It does not follow, however, that

these are causally related, for dogs in which Goldblatt and his co-workers had produced marked persistent hypertension by constriction of the renal arteries retained a normal urea concentrating power, and some dogs in which hyposthenuria had been produced by subtotal nephrectomy showed no significant rise in blood pressure.

While it might be possible to explain the increased volume of dilute urine passed in chronic Bright's disease on the basis of flood diuresis resulting from the elevated capillary pressure and possibly increased blood flow through the remaining nephrons, the facts could be accounted for equally well if the remaining tubular epithelium had lost its concentrating power. The evidence on this point is indirect. If about half of one kidney is removed from a dog, and some weeks later the other kidney is excised, the animal puts out an increased volume of dilute urine, and the specific gravity is not raised significantly by the withholding of water for twenty-four or even forty-eight hours.[4] Kidney function is reduced, blood urea level is elevated, and in some animals blood pressure is moderately elevated. With a reduced number of nephrons, lowered urea clearance and apparent loss of concentrating power, the renal function of these animals simulates that of chronic nephritis. Injections of pituitrin and adrenal cortical extract, and the administration of a diet rich in protein had no effect on the specific gravity of the urine. When, however, the concentration of plasma protein was raised by severe dehydration or by the injection of concentrated plasma, the animals excreted a small volume of urine with a specific gravity as high as during the control period before operation. The increase in concentration of plasma protein presumably diminishes effectual pressure in the glomerular capillaries, resulting in a smaller volume of glomerular filtrate and consequent better opportunity for reabsorption of water by the tubular epithelium. Urine of high specific gravity was also obtained when blood pressure was lowered to just above the critical level by spinal anesthesia. Here again, the mechanism is probably dependent on a smaller volume of filtrate. Since it is hard to believe that either of these procedures would suddenly restore abnormal tubular epithelium to normal, it seems fair to assume that the hyposthenuria was due to changes in blood pressure or flow, or to changes in the composition of the blood, rather than to abnormality of the tubule cells. A few observations of the effect of lowered blood pressure on patients suffering from chronic nephritis are consistent with this. Both Christian and Mosenthal have commented that a patient who has had hyposthenuria for a long period may excrete urine of high specific gravity before death. I have confirmed this finding, and found that it occurs when systolic blood pressure falls to 70 to 90 millimeters of mercury and is maintained at this level for some hours. Patients with systolic blood pressures of 150 to 220 millimeters of mercury who did not excrete urine of higher specific gravity than 1.009 and 1.010 (corrected for any protein present), have, when blood pressure fell to near the critical level, excreted urine having a specific gravity of 1.015 or 1.018. No opportunity has yet been presented to study the effect of increased concentration of plasma protein. Pituitary extract is without effect on the patient suffering from chronic nephritis, as it is on the dog with subtotal nephrectomy.

The composition of the urine, that is the contribution to the total specific gravity of urea, chloride, sulphate, et cetera, is usually the same in chronic nephritis as in the normal subject. It is simply more dilute.

SUMMARY

Chronic nephritis, including glomerulonephritis, arteriolar nephrosclerosis, and pyelitis, is characterized by a reduction in the number of kidney units or nephrons. Large numbers of destroyed glomeruli may completely disappear, without leaving recognizable scars.

Kidney function, as measured by the urea clearance or maximum specific gravity test, correlates better with the number of remaining nephrons than it does with kidney weight. While qualitative changes in the

tubular epithelium of the remaining nephrons cannot be excluded, it seems probable that the characteristic increased volume of dilute urine in chronic nephritis may be in the nature of a flood diuresis through a small number of nephrons, mediated through changes in the composition of the blood, in the calibre of vessels, or in blood flow.

The progressive destruction of nephrons in all of the more common types of chronic nephritis accounts for the similarity of symptoms in the late stages. A differential diagnosis may be impossible in the absence of an accurate history of the onset and course of the disease. Even the histologic appearance of the kidney may be less helpful than is commonly believed, since destroyed glomeruli completely disappear, and it is not justifiable to assume that those remaining necessarily show the same type of pathologic process.

BIBLIOGRAPHY

1. Kunkel, P. A.: The number and size of the glomeruli in the kidney of several mammals, Bull. Johns Hopkins Hosp., 47:285, 1930.
2. Moritz, A. R., and Hayman, J. M., Jr: The disappearance of glomeruli in chronic kidney disease, Am. J. Path., 10:505, 1934.
3. Hayman, J. M., Jr., Martin, J. W., and Miller, M.: Renal function and the number of glomeruli in the human kidney, Arch. Int. Med., 64:69, 1939.
4. Hayman, J. M., Jr., Shumway, N. P., Dumke, P., and Miller, M.: Experimental hyposthenuria, J. Clin. Invest., 18:195, 1939.
5. Alving, A., and Van Slyke, D. D.: The significance of concentration and dilution tests in Bright's disease, J. Clin. Invest., 13:969, 1934.

THE TREATMENT OF ANURIA*

THOMAS FINDLEY, M. D.†

NEW ORLEANS

Every sensitive physician knows that invisible but powerful forces are at work in each sick room he visits. These forces, emanating from the patient and his family, constitute a demand for action on the part of the physician which may at times be almost irresistible. In an emergency this urge may be so great indeed that any action taken by the physician, no matter how aimless or harmful it may really be, is eagerly offered and gratefully received.

Abrupt cessation of urinary flow always presents an imperative call to action and there are many ways in which the physician may respond. Under current practices he is tempted to meet the challenge with the infusion needle or the scalpel through the renal capsule, but other and possibly saner courses of action are also open to him. He may, for example, arrive at the difficult decision that it is wiser to do virtually nothing at all.

It is conventional to deal with the problem of anuria first by offering a classification which is supposed to pigeonhole the various types in a clear-cut convenient form, but our knowledge of renal physiology is changing so rapidly that any rigid approach seems ill-advised. It is apparent, however, that anuria is always due to *glomerulotubular imbalance* and may thus be divided into two main categories.

ANURIA OF GLOMERULAR ORIGIN

Diminution in the rate of glomerular filtration is due to reductions in the rate or pressure of glomerular blood flow. Hypofiltration and oliguria are therefore constant features of any circumstance which deviates blood from the kidneys, and the renal vasoconstriction which accompanies peripheral circulatory failure from any cause may be so intense as to produce anuria and permanent renal damage.[1-4] The reduced urinary volume which follows severe physical exertion, congestive heart failure, trauma, hemorrhage, burns, dehydration, the hepatorenal syndrome, and certain infections, is probably always initiated by renal ischemia. Prolonged ischemia causes tubular lesions which may not be reversible.

Recently, Trueta and his colleagues[5] described a phenomenon characterized by renal ischemia limited to the cortex which, if confirmed, may clarify certain discrepancies known to exist between renal blood flow and urinary formation. For many years observers have occasionally noted that red blood could be seen leaving the

*Read at the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, April 13, 1948, in Monroe, La.

†From the Departments of Medicine, Tulane University School of Medicine and the Ochsner Clinic, New Orleans.

kidney through the renal vein at a time when urinary formation was greatly diminished. Utilizing perfusion and radiographic technics, Trueta's group showed not only that the combination of cortical pallor, oxygenation of renal vein blood, and anuria could be produced by various means, but that this phenomenon was caused by the redistribution of blood from the cortex to the medulla of the kidney. Their explanation is based on certain anatomic differences between the familiar glomeruli in the more peripheral portions of the cortex and the less numerous *juxtamedullary* glomeruli in the more central portion of the cortex. The peripheral glomeruli through which the blood supply of the kidney normally flows are equipped with efferent arterioles which are relatively small in caliber and which lead by multiple branches into the vast system of peritubular capillaries; the efferent vessels of the juxtamedullary glomeruli, on the other hand, are much larger and they lead directly into the relatively capacious loops of the vasa recta and the interlobular veins. It is claimed that these medullary channels are large enough to accommodate the entire renal blood flow, and that the transfer of blood from the cortical to the medullary circuit may be produced by the action of certain hormones, by stimulation of the renal nerves, by hemorrhage and by tourniquet shock. Bilateral cortical necrosis has even been produced by the intravenous administration of staphylococcic toxin.

The implication of these observations is far-reaching. If blood entering the kidney through the renal artery can traverse the kidney by one of these two routes or by a combination of them in varying degree, the interpretation of renal clearance tests becomes difficult, and current concepts regarding the action of diuretic and antidiuretic drugs must certainly be revised. It must be noted, however, that most observers[4] have found the total renal blood flow in shock to be reduced rather than increased. It is too early to evaluate Trueta's claims accurately but it seems probable that this mechanism for redistributing blood within the kidney is not too important in the human at least.[6] The existence of these medullary channels has been known for many years. They undoubtedly offer less resistance to the flow of blood than do the cortical vessels but the circumstances under which they are called into play and the degree to which they are able to maintain total renal blood flow remain to be established.

Glomerular anuria is also occasionally encountered, of course, in acute glomerulonephritis, but this condition should be recognized by standard clinical methods. No means of altering the swelling and proliferation of the glomerular endothelium is known at the present period. Diuretics are obviously useless, and the ingestion of salt and water should be sharply limited until the vascular reaction subsides.

Treatment: Since the persistence of shock for only a few hours is apt to produce chronic renal failure, it is urgently important to combat peripheral circulatory collapse by any means which seem appropriate. Except in those rare instances in which shock is due to failure of the heart rather than to diminished blood volume, these will include prompt and adequate transfusion of whole blood. Since acidosis is not likely to be severe in the early phase of shock, the parenteral use of alkali in this stage is not usually needed. Even if hemoglobinuria indicates the existence of a transfusion reaction, the administration of base for the purpose of alkalinizing the urine is unlikely to accomplish this end and may produce severe metabolic alkalosis.[7] The use of high spinal anesthesia or splanchnic procaine block is not justified on the basis of available evidence.

ANURIA OF TUBULAR ORIGIN

Acute toxic nephrosis seems a less cumbersome term than "lower nephron nephrosis"[8] and is more accurately applied to those cases of anuria in which nephrotoxins may be suspected of playing a role. Since renal ischemia undoubtedly produces degenerative changes in the tubules, and since circulatory changes also undoubtedly complicate the picture of tubular poisoning, it

is often not possible to distinguish sharply between these two types of renal damage. Dehydration, electrolyte imbalance and shock only too commonly accompany any process apt to damage tubules directly.

Among those agents thought capable of producing direct degenerative lesions of the convoluted tubules the most frequent are anoxia, heme pigments (hemolytic crises), heavy metals (mercury, bismuth, arsenic, uranium), sulfonamides, carbon tetrachloride, alcohol, mushrooms and tissue extracts. All these probably diminish urinary volume by destroying the capacity of tubular epithelium for the selective reabsorption of glomerular filtrate because Richards[9] directly demonstrated that the glomerular circulation of frogs poisoned with bichloride of mercury is entirely adequate at a time when no urine is issuing from the ureter.

From the standpoint of prognosis it is important to remember that the renal tubules possess great capacity for regeneration and repair. Complete restitution of structure and function is to be expected if exposure to the noxious agent is not extraordinarily intense and if the patient survives the period of immediate dysequilibrium.

Treatment: It has been pointed out that anuria of glomerular origin quickly assumes the characteristics of that due to tubular dysfunction. Except, therefore, for the measures needed to prevent and combat early shock, treatment of both varieties should proceed along identical lines.

Under any circumstances the mortality rate will be high but it can be appreciably lowered if efforts are made to prevent death from circulatory failure. Recently some much needed figures concerning the effect of anuria and fluid administration upon the total circulating blood volume have been published;[10] in patients receiving more than 1000 cc. of fluid daily the average increase in blood volume was more than 40 per cent, and it was about 20 per cent even in those who were given less than this amount of liquid. These figures explain the high incidence of death from pulmonary edema and illustrate the dangerous futility of attempts to initiate diuresis by fluid administration. Estimates concerning the daily requirement of normal man for physiologic saline vary from 300 cc.[11] to 700 cc.;[12] a compromise suggests that anuric patients need no more than 500 cc. of water and 4 Gm. of sodium chloride daily, unless vomiting, sweating, or diarrhea increases the demand. It is far better, and usually possible, to give saline orally in the form of low protein, high carbohydrate beverages.* If edema appears, sodium should be entirely eliminated from the diet. This rigidly restricted intake of fluid must be continued until spontaneous diuresis occurs, at which time the volume of fluid given may be adjusted daily to equal the volume of urine excreted. Since the clinical crisis usually terminates in recovery or death in about ten days, total caloric consumption may be largely disregarded, as may moderate degrees of acidosis and hypochloremia.

Sharp reductions in plasma carbon dioxide and chloride content should, of course, be corrected. If one wishes to give sodium bicarbonate parenterally for this purpose, the nomogram of Van Slyke[1] is useful but the volumes of isotonic solution called for seem dangerously great for use in hydremic patients; a man weighing 170 pounds requires 6.5 liters of 1.3 per cent sodium bicarbonate to increase the plasma carbon dioxide from 15 volumes to 60 volumes per cent, if 0.026 Gm. of sodium bicarbonate per kilogram of body weight is calculated to raise the plasma carbon dioxide by 1 volume per cent. Whenever practical, it is safer to give sodium citrate orally in doses of 20 to 30 Gm. daily until the deficiency of base is met; if infusion is needed, sodium bicarbonate or sodium lactate may be given slowly in hypertonic solution (1-3 to 1-2 molar) in order to re-

*Miss Corinne Baker, Chief Dietitian of the Ochsner Clinic, devised the formula: 240 cc. medium cream, 2 eggs, 25 Gm. lemon juice, 100 Gm. lactose, one-half teaspoon sodium chloride, 0.5 cc. Vi-penta Drops (Roche). It is not unpalatable and supplies 20 Gm. protein and 1064 calories.

duce fluid intake. Molar sodium lactate in a dose of 10 cc. per kg. will increase the plasma carbon dioxide content by approximately 33 volumes per cent, an amount great enough to relieve the clinical signs of acidosis.

Urologists appear to rely heavily upon the intravenous use of sodium sulfate but it would be difficult to devise a remedy more likely to induce hydremia. Except in the presence of severe acidosis all fluids containing large amounts of sodium should be strictly prohibited, and this is one of the objections to transfusion since each unit of whole blood contains about 2 Gm. of sodium citrate.[7] Transfusion may properly be used early in an effort to combat shock but red cells suspended in sodium-free solutions might be less disturbing if anemia requires treatment in the later (twelve to twenty-four hours) stages. The average patient will more often require venesection than transfusion, however, since this is the most certain way of relieving the heart overburdened by hypervolemia.

With the exception of analgesics and hypnotics there are no drugs useful in the treatment of anuria. Digitalis and aminophyllin lower venous pressure and thus improve the output of an overloaded heart[13] but, in the absence of adequate renal function, are far less effective than bleeding in the management of pulmonary edema.

Nothing which is known about the physiology of the anuric kidney justifies decapsulation. Although there is no reason to deny that interstitial edema and cellular infiltration may accompany the acute nephrotic lesions seen in anuria, there is remarkably little information available concerning intrarenal pressure either in health or disease. Surgeons who decapsulate kidneys in the expectation of increasing urinary flow usually justify themselves by assuming that the operation either diminishes intrarenal pressure or that it denervates renal blood vessels. The latter hypothesis is manifestly untenable but the former theory may sound plausible because of numerous clinical assertions that anuric kidneys often look swollen to the naked eye and that capsulotomy results in bulging of the parenchyma through the incision. In a recent review of this subject we[14] were unable to find any recorded instances in which intrarenal pressure had been actually measured in man and, even more remarkable, only one attempt to make such a measurement in normal animals.[15] We agree with Kugel[16] that gross renal edema is probably due to the administration of the excessive amounts of fluid in such patients, and are inclined to place much more confidence in the descriptions of anuric human kidneys which were not swollen and which did not bulge after stripping of the capsule. A survey of the literature on decapsulation over the past twenty years allowed us to conclude only that surgeons do not often report their failures and that fewer patients die after unilateral than after bilateral decapsulation.

Although still in the experimental stage and unavailable to the profession at large, the "artificial kidney" promises to be of real aid in supporting the patient through the critical period required for tubular regeneration.[17-19] Peritoneal dialysis presents a formidable technical problem largely because of infection,[20-22] and irrigation of the small intestines through a three-lumened tube has not yet reached the stage of clinical application.[23]

CONCLUSIONS

1. Restriction of water and sodium intake is essential in the treatment of anuria if the mortality rate from circulatory failure is to be reduced.
2. Parenteral administration of fluid is potentially dangerous and seldom necessary.
3. Chemical denervation of the kidneys is not of proved value, and renal decapsulation is condemned.

REFERENCES

1. Lauson, H. D., Bradley, S. E. and Cournand, A.: Renal circulation in shock, J. Clin. Investigation, 23:381, (May) 1944.

2. Phillips, R. A. *et al.*: Effects of acute hemorrhagic and traumatic shock on renal function of dogs, Am. J. Physiol., 145:314, (Jan.) 1946.

3. Selkurt, E. E.: Renal blood flow and renal clearance during hemorrhagic shock, Am. J. Physiol., 145:699, (March) 1946.

4. Van Slyke, D. D.: Effects of shock on the kidney, Ann. Int. Med., 28:701, (April) 1948.

5. Trueta, J., Barclay, A. E., Daniel, P. M., Franklin,

K. J. and Prichard, M. M. L.: Studies of the Renal Circulation, Springfield, Ill., C. C. Thomas, 1947.

6. Reubi, F. C., Schroeder, H. A. and Williams, A. H.: Are there arterio-venous shunts in the kidney? Federation Proc., 7:101, (March) 1948.

7. Burnett, C. H. *et al.*: IV. Effects and use of alkalies in traumatic shock, Surgery, 22:1029, (Dec.) 1947.

8. Lucké, B.: Lower nephron nephrosis (renal lesions of crush syndrome, of burns, transfusions, and other conditions affecting lower segments of nephrons), Mil. Surgeon, 99:371, (Nov.) 1946.

9. Richards, A. N.: Direct observations of change in function of renal tubule caused by certain poisons, Tr. A. Am. Physicians, 44:64, 1929.

10. Burnett, C. H., Shapiro, S. L., Simeone, F. A., Beecher, H. K., Mallory, T. B., and Sullivan, E. R.: III. Post-traumatic renal insufficiency, Surgery, 22:994, (Dec.) 1947.

11. Burnett, C. H.: The clinical and therapeutic aspects of acute renal insufficiency due to lower nephron or hemoglobinuric nephrosis, Am. Pract., 2:6, (Sept.) 1947.

12. Butler, A. M. and Talbot, N. B.: Medical progress; parenteral-fluid therapy; estimation and provision of daily maintenance requirements, New England J. Med., 231:585, (Oct. 26) 1944.

13. Howarth, S., McMichael, J. and Sharpey-Schafer, E. P.: The circulatory action of theophylline ethylene diamine, Clin. Sc., 6:125, 1947.

14. Culpepper, W. S. and Findley, T.: Renal decapsulation for oliguria and anuria, Am. J. M. Sc., 214:100, (July) 1947.

15. Brodie, T. G.: Croonian Lecture: A new conception of the glomerular function, Proc. Roy. Soc., London, (Series B), 87:571, 1914.

16. Kugel, V. H.: Management of acute toxic nephrosis, Am. J. Med., 3:188, (Aug.) 1947.

17. Kolff, W. J.: The artificial kidney, Holland, Kampen, 1946.

18. Murray, G., Delorme, E. and Thomas, N.: Development of an artificial kidney, Arch. Surg., 55:505, (Nov.) 1947.

19. Alwall, N., Norvilt, L. and Steins, A. M.: Clinical extracorporeal dialysis of blood with artificial kidney, Lancet, 1:60, (Jan. 10) 1948.

20. Fine, J., Frank, H. A. and Seligman, A. M.: Treatment of acute renal failure by peritoneal irrigation, Ann. Surg., 124:857, (Nov.) 1946.

21. Abbott, W. E. and Shea, P.: Treatment of temporary renal insufficiency (uremia) by peritoneal lavage, Am J. M. Sc., 211:312, (March) 1946.

22. Bassett, S. H. *et al.*: Nitrogen and fluid balance in treatment of acute uremia by peritoneal lavage; analysis of peritoneal washings for protein, nonprotein nitrogen, and phosphorus, Arch. Int. Med., 80:616, (Nov.) 1947.

23. Rogers, J. W., Sellers, E. A. and Gornall A. G.: Intestinal perfusion in the treatment of uremia, Science, 106:108, (Aug. 1) 1947.

THE PRACTITIONER AND THE DIAGNOSIS AND TREATMENT OF HEADACHE*

WILLIAM WALLACE MORRISON, M. D.

NEW YORK, N. Y.

The subjective symptom of headache is perhaps the most common complaint of those who seek help from the practitioner. Headache is, of course, caused by very many physical and mental disturbances. In a few types of headache the experienced physician will be able to make a correct diagnosis on the basis of a simple history and a brief, local physical examination. As we shall see, however, with many other kinds of headache such a course can readily lead to serious mistakes in diagnosis with consequent failure in treatment.

It is therefore evident that there is need for a careful, systematic diagnostic study of frequently recurring or chronic but severe headaches which temporarily or more permanently disable the patient. If the practitioner will devote the necessary time and effort to such a diagnostic study, many headache patients will avoid the necessity of going from doctor to doctor before securing relief.

We will exclude consideration of the simpler headaches which form only a minor part of many symptom complexuses of obvious cause and which are usually either mild or very temporary, and devote our time to a study of the cases in which headache is the chief symptom, or at least is one of the most marked complaints.

We will also exclude any mention of the several types of neuralgia of the head and neck. These are usually fairly localized pains, often of characteristic type, which are chiefly of purely nerve origin and distribution, in contrast to the more generalized head pain which we term headache.

THE PAIN-SENSITIVE STRUCTURES OF THE HEAD AND NECK

Recent studies of the pain-sensitive structures of the head and neck and of the mechanisms of production of a number of types of serious headache, made chiefly by Wolff and his associates, are of very great interest, and aid importantly in our understanding of the diagnosis and treatment of headache. They form the basis of a part of a diagnostic plan by which headaches may be studied.

These investigators made repeated and careful examinations to determine which of the structures of the head and neck are actually pain-sensitive. They studied par-

*Read at Eleventh Annual Meeting of the New Orleans Graduate Medical Assembly, February 25, 1948.

ticularly the intracranial structures which had been exposed during various neurologic operations under local anesthesia, for sensitivity to various kinds of stimuli.

The essentials of their many reports are that (1) the tissues covering the cranium and adjacent to it are all more or less sensitive to pain; the arteries and nerves are especially sensitive. The structures of the face, neck and organs of special sense are all quite sensitive to pain. (2) The cranium itself including the diploic and emissary veins, the brain parenchyma, most of the dura, most of the pia-arachnoid, the ependymal lining of the ventricles and the choroid plexuses are all insensitive to pain. (3) Of the intracranial structures, the great venous sinuses and their tributaries from the surface of the brain, parts of the dura at the base of the brain, the dural arteries, and the cerebral arteries of the base of the brain, and the fifth, ninth, and tenth cranial nerves as well as the upper three cervical nerves are all sensitive to pain.

THE MECHANISMS OF HEADACHE

Wolff states that headaches may be divided into two main categories: those that arise mainly as a result of stimulation of pain-sensitive intracranial structures, and those occurring chiefly by stimulation of pain-sensitive structures on the outside of but adjacent to the skull. He points out that intracranial disease usually produces referred pain. Wolff clearly showed that stimulation of pain-sensitive structures on or above the tentorium cerebelli results in pain in front of a line drawn across the top of the head just in front of the ears. The pathways for the referral of such pain are contained within the fifth cranial nerve, so that it is felt over the area supplied by one or more of the divisions of the trigeminal nerve. Stimulation of pain-sensitive structures below the tentorium results in pain behind the line described. The pathways for the referral of this pain lie chiefly within the ninth and tenth cranial nerves and the upper three cervical nerves.

Six basic mechanisms have been postulated by Wolff and his associates as a result of their studies. These are that headache results from:

1. Traction on veins passing to the great venous sinuses within the skull, and by displacement of the sinuses themselves.
2. Traction on the middle meningeal arteries.
3. Traction on the large arteries of the base of the brain and their main branches.
4. Distention and dilatation of the intracranial arteries.
5. Inflammation in or about any of the pain-sensitive structures of the head and neck.
6. Direct pressure as by tumors on the cranial and cervical nerves which contain pain-afferent fibers for the head and neck, which have been named.

These authors point out that intracranial disease usually produces headache through more than one of these mechanisms acting at the same time, and by involvement of more than one pain-sensitive structure.

Wolff emphasizes that traction on, displacement, distention and inflammation of cranial *vascular* structures are chiefly responsible for the production of headache. This is in direct contradistinction to older beliefs that head pain, particularly intracranial headache was caused by changes in *nerve* structures almost exclusively.

THE PATHOLOGIC PHYSIOLOGY OF HEADACHE

Pfeiffer and his associates have pointed out that a disturbance in the hydrodynamic mechanism within the cranium accounts for the production of a majority of types of headache. The pain-sensitive dura, falx, tentorium and cranial blood vessels may be distorted by factors which disturb the normal relation between the brain volume, the cerebrospinal fluid pressure, the blood volume and the peripheral vascular tone.

Thus the brain volume is altered by tumors and abscesses, and possibly by toxins as in uremia to cause headache. The cerebrospinal fluid pressure is increased in spinal blocks as in meningitis, and in carbon monoxide poisoning, and decreased after spinal drainage, with headache. The blood volume is increased by such drugs as the nitrites and histamine, and is decreased in migraine, with headache. Peri-

pheral vascular tone is increased in hypertension and probably decreased in migraine with headache.

THE RELATIVE FREQUENCY OF THE CAUSE OF HEADACHE

Although no two authors on the subject use quite the same classification, or agree fully as to the relative frequency of the various causes of disabling headache, the following order of frequency, comprising the ideas of Wolff and Moench, is one which we can use as a basis for our consideration of the individual types of etiology, so that we may discuss the diagnosis and treatment in orderly fashion.

1. Psychogenic headache of emotional origin; Moench considers this to be the most common of all causes for headache; we will consider it last.

2. Migraine.

3. With pathology in the structures of the neck, either sustained muscle spasm, now generally called myalgia due to a variety of causes, or to actual arthritis of the cervical spine.

4. With systemic disease with fever or toxemia or hypertension.

5. In nasal and paranasal sinus disease.

6. In ocular disease.

7. With intracranial pathology.

8. Histamine headache.

MIGRAINE

Typical, true migraine is thought to be the most common cause of temporary if recurrent incapacity due to headache. It has been estimated that as many as eight million people in the United States suffer from migraine in mild or severe form. It usually starts before the age of thirty-five years and may be seen in children. It tends to affect the upper class of educated and intelligent people most often. Heredity plays a role in 50 per cent of the cases and there is a definite allergic background very often.

It is a periodic headache, usually unilateral at onset, but becoming more generalized later. It is associated with irritability, nausea and vomiting, constipation or diarrhea, and in about 10 per cent of cases is ushered in by "fortification" scotoma, photophobia or hemianopsia. Each attack lasts for several hours to several days and there may be euphoria between seizures.

Wolff and his associates have demonstrated the actual mechanism of the pain in true migraine to be an increase in the amplitude of the pulsations of the extracranial arteries, chiefly the branches of the external carotid artery, by vasodilatation and consequent stretching. The pain is thus extracranial and vascular in origin. Wolff believes that in addition there is prolonged contraction of the muscles of the neck and scalp as in myalgia, along with a transient edema of the walls of the distended carotid arteries. He considers that these two factors prolong the headache after the vasodilatation passes, and explain the failure of relief seen when vasoconstrictor medication is given late in the attack. He believes the cause of the prodromal scotoma and other cerebral changes to be an early and very transient vasoconstriction of cerebral vessels.

The diagnosis of the typical form of true migraine rests on the presence of what Moench considers to be four classic components, any one of which may predominate or be absent. These are (1) headache, typically a severe hemicrania; (2) gastrointestinal disturbance, usually with nausea and vomiting; (3) visual disturbance preceding the pain, the scintillating scotoma being characteristic; and (4) history of a familial tendency to the same type of headache, very often with an allergic background. With these findings one can make a diagnosis of true migraine with reasonable certainty. (Table 1).

TABLE 1. DIFFERENTIAL DIAGNOSIS OF HEADACHE (Modified from Moench)

	MIGRAINE	MYALGIA	CERVICAL ARTHRITIS	NEPHRITIS, UREMIA	HYPERTENSION	HISTAMINIC HEADACHE
Frequency	Very common; Women 2.5; 1 men	Very common	Fairly common	Not common	Common	Rare, in males
Age	Puberty to menopause; absent in pregnancy	Adults	Usually over 45	Any	Usually over 40	Over 50 years
Type	Always periodic	Periodic	Constant	Constant	Periodic	Always periodic
Onset	Abrupt or gradual	Gradual	Gradual, increasing	Slowly over days	Gradual over months	Very abrupt
Duration	Hours to days	Variable	Long lasting	Long lasting	Hours	Less than one hour
Location	Hemicrania, may become bilateral	Back of neck into scalp	Neck into occiput and shoulders	General over head	Fronto-occipital	Temporal hemicrania
Character	Boring, throbbing, mild to severe	Aching with stiffness	Steady with soreness and stiffness	Dull	Throbbing	Excruciating, boring
Time	Any, especially with menses	Seldom wakens	Early morning	Any	Daily, wakens in morning	Wakens after short sleep
Posture and Activity	Worse lying down	Comfort with head held in hands	Cough, strain, use aggravate	No effect	Disappears after rising	Erect posture often relieves
Occupation	Professional, mental workers	Holding head in same position	Use of neck and shoulders	Any	Any	Any
Personality	Rigid	Rigid, neurotic		Any	Rigid, tense	Any
Light, Reading Noise, Travel	Aggravate pain	Aggravate pain	No effect	No effect	May increase	No effect
Lacrimation, Photophobia	May be present	None	None	None	None	Unilateral common on side of pain
Nausea, Vomiting	Nearly always	None	None	Very common	Very rare	Very rare
Myalgia of neck as complication	Quite common if pain prolonged		May be present	None	May occur	None
Miscellaneous	Scotoma; familial tendency; may be allergic history	Tender, stiff neck and scalp muscles	Crepitus; positive spinal X-rays	Signs of causative disease	Elevated blood pressure, etc.	Flushed skin, rhinorrhea, tenderness of carotid artery on same side

Certain diagnostic tests have been developed which serve to aid materially in establishing the presence of true migraine and in differentiating it from migranous headaches due to several causes which we shall consider in a moment. During the pain, inhalation of amyl nitrite will bring on a sharp increase in the discomfort. In the intervals between attacks, 1/50 of a grain of nitroglycerine under the tongue will cause a brief hemicrania in migraine subjects. During the attack, inhalation of pure oxygen for a time will often relieve the pain. Most characteristic is the effect of a hypodermic of ergotamine tartrate, 0.25 to 0.5 cubic centimeter, which usually brings prompt, often dramatic relief of pain. (Table 4).

TABLE 4. DIAGNOSTIC TESTS USEFUL IN DIFFERENTIATION OF MIGRAINE

IN INTERVAL BETWEEN ATTACKS:	
Histamine injection	May cause brief headache
Amyl nitrite inhalation	May cause very brief headache
Nitroglycerine 1/50 grain under tongue	Will often cause headache
DURING ACUTE ATTACK:	
Ergotamine injection 0.25 to 0.5 cc.	Will usually stop pain
Trichlorethylene inhalation	Will often stop pain for a time
Intravenous epinephrine 0.1 cc. 1/40,000	May stop pain
Inhalation 100 per cent oxygen	May stop pain

Typical migraine may be mimicked by at least three types of migranous headache. These occur (1) with hypertension, (2) with organic disease of the central nervous system, and (3) as headaches of emotional origin. All three of these may be marked by attacks of headache, often a hemicrania, but the other characteristics of true migraine are present in minor degree if at all. The diagnostic tests described usually give equivocal results and aid greatly in differentiation; the use of ergotamine in particular usually has little or no effect on migranous head pain.

These migranous headaches will be discussed under their appropriate headings. That they occur quite frequently, give rise to diagnostic problems and require quite different treatment from true migraine, clearly points to the need for the diagnostic study already advocated.

The actual cause of the vasodilatation of the cranial arteries which produces the headache in true migraine is probably most often an allergy, nearly always to one or more foods. The diagnostic study of true migraine hence should include skin tests, avoidance of suspected foods, or elimination diets.

The treatment of true migraine lies well within the province of the practitioner. The most important part of the treatment of the acute attack is the use of vasoconstrictors such as ergotamine tartrate by hypodermic or intravenous injection. As early in the attack as possible 0.25 to 0.5 cubic centimeters are given and repeated if the pain recurs. Tablets of 1 milligram by mouth are not nearly so effective. Ergovinine and dihydroergotamine are newer preparations with similar action. The treatment of the underlying allergy is necessary to prevent recurrence of the attacks. This is accomplished by elimination of foods causing symptoms, and the use of the antihistamine drugs.

HEADACHE WITH PATHOLOGY IN THE NECK

One of the most common causes of headache is hypertonicity of the posterior neck and scalp muscles, now usually termed *myalgia*. Pain in the back of the neck and into the head may also be caused by cervical *spinal arthritis*, by inflammatory *fibromyositis* of the nuchal fascia and muscles, and by occipital *neuralgia*.

Myalgia is the most common cause. It occurs as a primary condition of an occupational nature in those whose work requires that they keep the head and neck in fixed positions for long periods as in reading, writing and typing, accounting, sewing, etc. Wolff and his associates have also clearly demonstrated by means of myo-

grams of the posterior neck and scalp muscles that sustained contraction occurs in histamine headache, in spinal drainage headache, with irritation of the conjunctiva, and in prolonged contraction of the extraocular muscles, in painful nasal sinusitis, and even in migraine, and that it is especially common in the tension headache of emotional or psychoneurotic origin. Myalgia is thus a common contributing cause to headache of other primary origin. One could readily term this form of it a reflex myalgia.

In myalgia there is pain, soreness and tightness due to actual spasm in the back of the neck and scalp, with tender spots and local indurations on palpation of the trapezius and other muscles. The actual cause of the painful spasm and induration in the muscles in the absence of any reflex causes has been thought by Williams to be due to physical allergy, as to cold, associated with the formation of excess histamine and with prolonged vasoconstriction in the muscles, and the pain is carried by sympathetic nerve tracts.

The diagnosis is made by palpating the tender and indurated muscles of the neck and scalp. The pain can be very rapidly relieved by the injection of 1 per cent procaine into the point of greatest tenderness. This constitutes a diagnostic test which not only clearly points to myalgia as the source of the headache, but is of marked therapeutic value as well. (Table 1).

The treatment of myalgia comprises local heat and massage to the posterior neck muscles, with the procaine injection mentioned. Collar immobilization may be required in resistant cases, and a change in posture and work habits is usually necessary. When the hypertonic muscles are due to a reflex from the other sources mentioned they must be treated; psychotherapy directed toward the relief of physical and psychic tension is particularly important. With the idea that physical allergy plays a part in the cause, vasodilator drugs such as nicotinic acid and the antihistaminic agents may be used in an effort to prevent recurrence of the condition.

Neck and head pain, usually occipital, is seen with arthritis of the cervical spine in middle and older ages. The pain in the head, neck, and shoulder is aggravated by marked motion of the spine and by straining or coughing. There is local stiffness, tenderness, pain on stretching, and sometimes crepitus; the diagnosis is confirmed by radiographs of the cervical spine. The treatment is similar to that for arthritis of other areas, and is well managed by the practitioner.

HEADACHE WITH SYSTEMIC DISORDERS

Headache is a symptom of many systemic disorders; it may be merely a mild annoyance or be a signal of serious disease. Wolff does not consider that pain in the head due to disease elsewhere in the body can occur, except with fever and hypertension and in rare cases of angina pectoris which have pain in the neck. He could not demonstrate other mechanisms which he could believe might cause headache. Moench, from the standpoint of the internist, calls attention to many systemic conditions with headache, in which actual cause and mechanism are often obscure. He believes that myalgia, variations in blood volume and in intracranial vascular tension may readily play significant roles in the production of these headaches.

Moench lists some of the more common causes of headache in systemic disorder as (1) alkalosis in alkaline medication for gastric ulcer; (2) with fever, in which Wolff and his associates have shown that the mechanism is like that in histamine headache and is due primarily to distention of intracranial arteries; it is seen in many acute infections, in sepsis and bacteremia; (3) glomerulonephritis, probably because of toxic metabolites or disturbance of intracranial hydrodynamics; (4) in uremia, possibly due to hypertension, disturbance of intracranial circulation, direct meningeal irritation or actual edema of the brain; (5) in arterial hypertension in which Wolff and his co-workers have demonstrated that the mechanism is like that in migraine, and

pain arises chiefly by dilatation and distention of certain branches of the external carotid arteries; he points out that this type of headache usually bears no direct relationship to the level of the blood pressure or to the pulse pressure although the phenomena are related; (6) in hypoglycemia when the blood level reaches 60 to 90 milligrams per cent; (7) in cerebrospinal syphilis. He points out that either organic or functional gastrointestinal disturbances rarely cause headache despite wide belief to the contrary. He also considers that constipation is a rare cause of headache; there is a large psychogenic factor in headache associated with constipation, conditioned by training and present day propaganda for laxatives; the old idea of auto-intoxication has been largely abandoned as unproven. Emotional tension usually leads to sympathetic overaction with parasympathetic inhibition, and spastic constipation is an incidental; headache also arises because of the psychic disturbance.

Migranous headache seen with hypertension is probably largely based on psychic tension. Moench points out that both hypertension and migraine tend to occur in the same type of tense, rigid personalities, with a similar hereditary factor, and that the mechanism in both is dilatation of the external carotid arteries and branches, though this is of quite different origin. Vasoconstrictor drugs relieve true migraine, but make hypertension headache worse; they should be used with great caution in the latter disease. Vasodilator drugs tend to relieve hypertension head pain, and make migraine pain worse; these factors are important in differential diagnosis.

The diagnosis of the cause of headache in these systemic conditions is made on the history, the physical findings and necessary laboratory studies; the need for systematic study is of course obvious (Table 1). The treatment lies entirely within the province of the practitioner and must be directed toward the cause of both headache and systemic disease.

HEADACHE IN NASAL AND PARANASAL SINUS DISEASE

In contradistinction to the belief of most layman and physicians "sinus" is not a common cause of frequently recurrent or chronic disabling headache. When it does occur it is in acute sinusitis; chronic, uncomplicated sinusitis rarely causes serious headache. Headache from frontal sinusitis is localized over the forehead, that of maxillary sinusitis is in the cheek and upper teeth, and that of sphenoid and ethmoid sinusitis is felt behind the eyes and in the vertex. Pain in the back of the head, the neck and into the shoulder is caused by sustained muscle spasm or myalgia, as a reflex from painful sinusitis.

The mechanism of nasal and sinus pain has been investigated by McAuliffe, Goodell and Wolff by applying painful stimulation without anesthesia in human volunteers and and during operations under local anesthesia, to the nasal and paranasal structures, plotting the sensitivity and reference of the pain. They found that the mucosa of the approaches to the sinuses such as the ostium of the antrum and the nasofrontal duct were highly sensitive, the turbinates and upper parts of the nasal cavity less sensitive, and the mucosa within the sinuses of low sensitivity. Most of the pains were referred over the second division of the trigeminal and less over the first division.

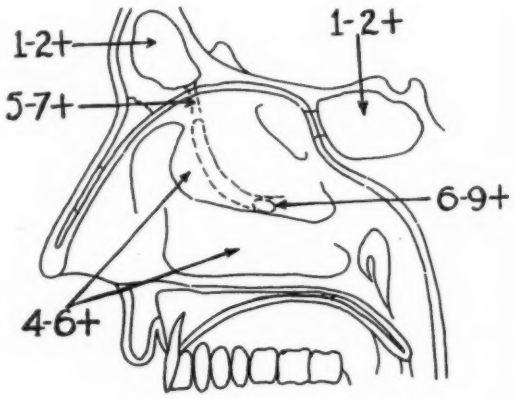


Figure 1. The relative pain-sensitivity of the nasal and sinus mucosa (McAuliffe, Goodell and Wolff).

They conclude that inflammation with engorgement of the turbinates, the superior nasal spaces and the sinus ostia are responsi-

TABLE 2. DIFFERENTIAL DIAGNOSIS OF HEADACHE (Modified from Moench)

	ACUTE SINUSITIS	CHRONIC SINUSITIS, VASOMOTOR RHINITIS	REFRACTIVE ERRORS, MUSCLE IMBALANCE	GLAUCOMA, IRITIS, ETC.	EMOTIONAL TENSION
Frequency	Not as common as supposed	Not very common	Quite common	Common when disease is present	Most common cause of headache
Age	Any	Any	School age, young adults and over 45	Any, usually adults	Usually adults
Type	Periodic daily	Periodic or constant	Periodic	Constant	Periodic or constant
Onset	After head cold	Gradual	With use of eyes	Usually gradual	Any type
Duration	Hours to end of day	Days to weeks	Hours, absent after sleep	May be acute or chronic	Hours, days, months, years
Location	Over involved sinus; referred to temple or vertex only	Frontal, temporal, orbital	Frontal, temporal, about or in eyes	In eye, frontal, temporal	Any, usually fronto-occipital and bilateral
Character	Throbbing	Dull ache, pressure	Dull, slowly increasing	Dull to severe, localized	Pain, tightness, feeling of band, cap, pressure, etc.
Time	Begins after rising, subsides afternoon	During day	Begins or increases at end of day	Worse in night	With and after emotional states
Posture and Activity	Stooping, jarring sharply aggravate	No effect	No effect	Aggravate pain	No effect
Occupation	Any	Any	Using eyes	Any	Any
Personality	Any	Any	Any	Any	Tense, anxious, psychoneurotic
Light, Reading, Noise, Travel	Aggravate	No effect	Distinctly aggravate	Aggravate	Aggravate, but diversion helps
Lacrimation, Photophobia	Usual, mild, with severe pain	None, except due to allergy	Usual but mild	Mild to severe	Weeping only
Nausea, Vomiting	Never	None	Very rare	With severe pain	May occur
Myalgia of neck as complication	Very common, causes occipital and neck pain	Uncommon	Very common, causes occipital and neck pain	May occur if pain prolonged	Almost the rule, causes occipital and neck pain and soreness
Miscellaneous	Respiratory infection precedes; pus in nose and sinuses	Allergic attack or head cold precedes	Errors of refraction or muscle imbalance found	Signs of eye inflammation, high intraocular tension	Many other symptoms and signs of psychoneurosis

ble for most of the pain in nasal and sinus infection and that headache not associated with these changes is probably not due to nasal or sinus disease. Zygomatic, frontal, cheek, temporal or vertex headache not greatly reduced in intensity or eliminated by application of vasoconstrictor and weak anesthetic medication within the nose is almost certainly not due to nasal disease. This constitutes a therapeutic test of great diagnostic worth.

The diagnosis of headache due to nasal and sinus disease is made on the history of a preceding acute upper respiratory infection, the presence of turbinate congestion and pathologic secretion on examination of the nose with the head mirror and reflected light, the results of transillumination of the frontal and maxillary sinuses and the results of shrinking and mild anesthetization of the nasal passages (Table 2).

Mild forms of nasal and sinus infection are readily treated by the practitioner with nasal vasoconstrictors, coal tar sedatives, local heat and chemotherapy. In severe, resistant or obscure cases the patient with headache due or probably due to sinus infection should be referred to the otolaryngologist.

HEADACHE DUE TO OCULAR DISEASE

Pain is often associated with intraocular disease such as glaucoma, iritis, choroiditis, etc.; it is usually localized at first, later spreading over parts of the area supplied by the first division of the trigeminal nerve. Such organic disease of the eye is readily discovered by careful external and internal examination of the eye, with a recording of the intraocular tension; simple palpation will serve the practitioner.

Headache is commonly believed to be due to "eye strain", and there is often headache associated with aniesokonia, excess accommodation or convergence, hypermetropia, astigmatism, muscle imbalance and unequal myopia, in which the pain is probably due to spasm and fatigue of the ciliary and/or extraocular muscles, analogous to myalgia. In many such cases, there is a large additional factor of myalgia of the posterior neck and scalp muscles from holding the head in a strained and fixed position, in efforts to produce distinct retinal images or binocular fusion. Wolff showed that both conjunctival irritation and extraocular muscle strain causes myalgia.

The diagnosis of headache due to these more obscure ocular conditions is made on the history of pain brought on by and increased with use of the eyes, located about the eyes, radiating to the brow or occiput as the day goes on, and usually bilateral and symmetrical. Local ocular symptoms and signs are also commonly present (Table 2). Exact diagnosis calls for a complete examination of the eye, and it is best to refer these patients to the ophthalmologist, since he will also supply the necessary treatment.

HEADACHE WITH INTRACRANIAL PATHOLOGY

Moench believes that intracranial disease causes about 5 per cent of all chronic headache, and that while the neurologic causes of such headache are few, they are important. Brain tumor causes about one death in every two hundred; no patient with persistent headache should be dismissed before the possibility of brain tumor has been carefully investigated.

The neurologic causes of headache are (1) brain tumor; (2) intracranial vascular disease such as thrombosis, embolism, hemorrhage, syphilitic arteritis and aneurisms; (3) intracranial inflammations such as meningitis, encephalitis and brain abscess; (4) post-concussion head injuries; and (5) a temporary form after spinal puncture, spinal anesthesia, ventriculography or encephalography.

The mechanism of headache in brain tumor, according to the investigations of Wolff and his associates is not increased intracranial pressure per se, but it is due to traction on or direct irritation of pain-sensitive intracranial structures, chiefly the large arteries, veins and venous sinuses of the brain, and certain cranial nerves.

TABLE 3. DIFFERENTIAL DIAGNOSIS OF HEADACHE (Modified from Moench)

	BRAIN TUMOR	MENINGITIS	BRAIN ABSCESS
Frequency	Causes 1 in 200 deaths	Uncommon	Quite uncommon
Age	Any	Any	Any
Type	Constant	Constant	Constant, varying
Onset	Gradual	Abrupt	Gradual
Duration	Prolonged	Prolonged	Very prolonged
Location	Local or referred	Occipital or generalized	Local or referred
Character	Dull or severe, boring	Throbbing	Dull ache
Time	Worse at night	Any	Any
Posture and Activity	Changes aggravate	Changes aggravate	May aggravate
Occupation	Any	Any	Any
Personality	May change	Acutely ill	May change
Light, Reading, Noise	Aggravate	Sharply aggravate	Aggravate
Lacrimation, Photophobia	May be present	May be present	Unusual
Nausea, Vomiting	Usual, may be projectile	Early and late	May occur late
Cranial nerve symptoms	May occur	Occur late	Occur
Papilledema	Common, late	Common, late	May occur late
Kernig sign		Strongly positive	
Fever, leucocytosis		High	High early and late
Spinal fluid changes	Occur	Very marked	Occur
Coma, somnolence	Late	Marked late	Usually late
Presence of suppurative focus		Very common	Very common

The diagnosis of probable brain tumor is made with a headache that is persistent and associated with repeated vomiting, and by the findings of a careful, routine neurologic examination. Findings will be uncovered which will indicate some intracranial lesion, and all such suspicious cases should be referred at once to the neurologist or neurologic surgeon for final diagnosis and treatment.

Intracranial vascular conditions causing headache will be discovered through the history, general physical and neurologic examinations, and the results of a spinal tap. These cases are usually very well treated by the practitioner. The diagnosis of meningitis is usually simple; when due to a suppurative focus, the patient should be referred to the otolaryngologist for surgical treatment; the practitioner can be responsible for necessary general care, with chemotherapy or antibiotic treatment. Brain abscess occurs with a demonstrable focus in the nasal sinuses or mastoid cells, after skull fracture and with lung abscesses or pyemia (Table 3). Such cases should be referred to the otolaryngologist, the neurologic surgeon or both.

Headache following spinal drainage is usually transitory. The mechanism has been demonstrated by Wolff and his co-workers to be traction on, or displacement of pain-sensitive structures of the brain by the lowered spinal fluid pressure. They point

out that greatly increased pressure is neither a prime nor essential factor in the production of headache.

Post-concussion headache is diagnosed on the history. It is probably due to localized intracranial vascular or perivascular changes following the trauma. There is very often a large psychic element, especially when litigation, compensation or psychoneurosis is a part of the picture. In the latter case part or all of the pain may be due to myalgia of the neck and scalp muscles, which can be demonstrated.

The pain of epidural or subdural hematoma and subarachnoid hemorrhage after trauma is due to local tissue damage and pressure, and the diagnosis and treatment of such conditions is best put in the hands of the neurologist.

A migranous type of headache may be noted in many types of intracranial disease. The symptoms are not typical of true migraine, and the neurologic findings will make the cause clear; the diagnostic tests for migraine will yield incorrect results.

HISTAMINE HEADACHE: HISTAMINIC CEPHALALGIA

This is a rare entity which produces a most characteristic headache. It is apparently due to accumulation of histamine in the body, because of either overproduction or failure of destruction in some as yet unknown manner. Sensitivity to cold and other allergy may play a part. Schumacher and Wolff have shown that it is due to vasodilatation with stretching of the cerebral arteries of the base of the brain, the internal carotid, the vertebral and basilar arteries, and the proximal segments of their main branches.

The diagnosis is made on the history of acute attacks of sharp headache in later life, lasting less than an hour, usually awakening the patient after a short sleep, strictly unilateral in the eye, temple, face or neck, usually with watering and congestion of the eye, nasal stuffiness, increased skin temperature with sweating and tenderness over the external carotid artery or its branches. The pain is quickly relieved in the erect position and made worse by lying down (Table 1).

The diagnosis is established by producing a typical attack of pain by the subcutaneous injection of from 0.1 to 1.2 milligrams of histamine. The pain produced is at once relieved by the intravenous injection of 0.1 cubic centimeter or more of 1 to 40,000 solution of epinephrine. The treatment under the supervision of the practitioner is gradual histamine desensitization and the use of antihistamine drugs.

HEADACHE OF EMOTIONAL ORIGIN

This type of headache is also called tension or relaxation headache, psychosomatic or psychoneurotic headache; head pain is rare in true psychoses. It is extremely common, being perhaps the most frequent cause of all the types of headache. It is noted in hypochondriasis, hysteria, anxiety and conversion neuroses, obsessive-compulsive states, in simple acute worry and in post-anxiety conditions. The apparent cause is usually a combination of worry, physical fatigue or excitement in psychoneurotic personalities. The mechanism of the pain is very largely that of nuchal and scalp muscle hypertonicity and hence is myalgic. Neck and scalp muscle spasm and indurations are the somatic evidence of the psychic tension and should always be sought for.

The diagnosis is suggested by the history. These head pains are nearly always capricious, bizarre, and without special pattern as to time, location, duration, or exciting factors. The nature of the pain is often characteristic, as it is often described simply as discomfort, pressure in the head, feeling of a tight band or cap, etc. The total duration is also characteristic; organic headaches do not last for years or a lifetime without physical accompaniments. Exaggeration of severity is the rule. There are always additional psychoneurotic complaints of dizziness, sweating hands, globus, precordial pain, dyspnea, stomach "trouble", and back or pelvic pain (Table 2).

The diagnosis of headache due to emotional tension or true psychoneurosis is made on the history, and the failure to find any reasonable physical cause for the pain. This, of course, demands that there be a full diagnostic study to search for causes, such

as true migraine or myalgia of the neck which produce some part of the symptom complex, and which are amenable to proper treatment. It is also important that serious brain disease such as tumor, or systemic disease be discovered by a complete study, for obvious reasons. Every effort should be made to rule out all other causes for headache before the diagnosis of psychic pain is made. Treatment is by psychotherapy.

Moench points out that migranous headache of purely emotional origin is fairly common; a differential diagnosis from true migraine is necessary. Attacks of severe hemicrania, with nausea are seen in psychoneurotics of high intelligence of great nervous drive, who are of the tense and worrisome type and who are under emotional strain. The actual attacks take place when periods of such tension are followed by sudden changes in mood or program. Pain may occur with an increase in tension and restraint as by criticism, failure, domestic crises, etc., or by release from tension, during relaxation as on a holiday. The attacks only superficially resemble true migraine and characteristically are accompanied by obvious signs of marked emotional imbalance.

Treatment of these attacks comprises the use of such vasodilators as nicotinic acid or histamine, which are of variable action, and the use of barbiturate medication to allay tension.

All serious emotional problems with headache are usually best handled by referral to the psychiatrist, unless the practitioner is trained in psychosomatic technic and can devote the necessary time to the treatment.

CONCLUSION

It is surely evident from this rapid survey of the problem that the diagnosis and proper treatment of chronic or recurrent headache is usually anything but a simple matter. As has been repeatedly noted, a careful study of the whole patient by means of a systematic history, complete physical examination, diagnostic tests, and at times trials of one or more therapeutic tests is essential. In this plan, the practitioner plays an important part. He can carry on much of the investigation and treatment, and serve as the co-ordinator for special studies which must be performed by specialists in some of the types of examination and treatment which most practitioners have not the time, equipment or training to do. He should be the key man in the study of headache.

With the view toward the orderly study of headache patients, an abbreviated outline for taking the history (Table 5), for making required physical examinations (Table 6), and for applying diagnostic and therapeutic tests is presented (Table 7). Much more complete information may be had by study of the publications of Wolff and his many associates and of Moench, in their books of recent issue.

REFERENCES

Wolff, Harold G.: Headache and other Head Pain, Oxford University Press, 1948.

Moench, L. G.: Headache, Year Book Publishers, Chicago, 1947.

TABLE 5. OUTLINE FOR HISTORY TAKING IN DIAGNOSIS OF HEADACHE

OBTAIN ANSWERS TO THE FOLLOWING QUESTIONS:

1. Is the headache the only complaint? What other complaints are there in order of occurrence, severity and duration?
2. Is headache the most important symptom?
3. Where is the pain usually located exactly?
4. Does the pain radiate? If so, in what direction and extent?
5. When did the headache first occur?
6. If periodic, what is the usual frequency?
7. What is the duration of the pain?
8. At what time of the day does the pain usually occur?
9. What is the character of the pain? How severe is it?
10. Does the pain come and go quickly or gradually?
11. Do any of the following repeatedly bring on the pain? Physical exertion or work, change in posture, heat, excitement, worry or disappointment, menstrual periods, use of alcohol, excessive smoking, damp or cold weather, exposure to drafts, nasal stuffiness, cold in the head, swimming, use of the eyes, any particular drink or food, constipation.
12. Do any of the following repeatedly occur just before or with pain? Spots before the eyes, impairment of vision, nausea or vomiting, ringing ears, dizziness, watering eyes, burning and redness of eyes, stuffiness and running of nose, tender spot in face, neck or scalp, stiff neck muscles, toothache, chill, malaise, fever, fatigue.

TABLE 6. OUTLINE OF PHYSICAL EXAMINATION IN DIAGNOSIS OF HEADACHE

THE PHYSICAL EXAMINATION SHOULD INCLUDE:

1. Recording of temperature, pulse and respiration rate and blood pressure
2. Examination of the head for local lesions, tender spots, tender suboccipital, pre- and postauricular lymph nodes
3. Examination of the neck for stiffness, tenderness, crepitus, spasm and induration of muscles, tender anterior and posterior lymph nodes
4. Complete examination of the eyes
5. Examination of the ears and mastoid processes
6. Examination of the nasal cavities and nasopharynx
7. Examination of the paranasal sinuses
8. Examination of the mouth, teeth, gums, tongue, pharynx, hypopharynx and larynx
9. General physical examination
10. Neurologic and/or psychiatric examination
11. Indicated laboratory tests such as urinalysis, blood count, Wassermann, blood chemistry, spinal fluid examination, etc.
12. Indicated radiographic examination of the skull, nasal sinuses, mastoids, teeth and jaws, cervical spine, etc.

TABLE 7. OUTLINE OF DIAGNOSTIC AND THERAPEUTIC TESTS IN THE STUDY OF HEADACHE

NOTE THE EFFECTS OF:

1. Inhalation of ampoule of amyl nitrite
2. Inhalation of ampoule of trichlorethylene
3. Nitroglycerine, 1/50 grain, under tongue
4. Prolonged inhalation of 100 per cent oxygen
5. Subcutaneous injection of 0.25 to 0.5 c.c. ergotamine tartrate
6. Subcutaneous injection of 0.1 c.c. or more of histamine
7. Intravenous injection of 0.1 c.c. of 1/40,000 epinephrine
8. Increasing doses of histamine by subcutaneous injection
9. Increasing doses of nicotinic acid by mouth or injection
10. Injection of 1 per cent procaine into tender spot in neck or scalp
11. Heat and massage to neck muscles
12. Change in work habits as to posture, lighting, etc.
13. Change in type of work, of living or work surroundings, or of climate
14. Use of nasal vasoconstrictor sprays or instillations
15. A trial of appropriate eye glasses
16. Elimination of suspected foods giving positive skin reactions or noted in food diary
17. Use of antihistaminic drugs
18. Proper treatment of any ocular, nasal, sinus, cervical spine, or neurologic condition found
19. Indicated specific treatment of any systemic disease found
20. Indicated symptomatic treatment of systemic disease when no specific exists
21. Psychotherapeutic treatment.

—o—

THE PRESENT STATUS OF THE FENESTRATION OPERATION FOR OTOSCLEROSIS*

WILLIAM WALLACE MORRISON, M. D.

NEW YORK, N. Y.

Fenestration for clinical otosclerosis is one of the truly outstanding advances in otology in the past ten years. By fenestration we mean the modern one-stage operation developed by Lempert, on the basis of earlier work by Holmgren and Sourdille, and first reported by him in 1937. In the intervening years, while the basic concept of the procedure has changed scarcely at all, a number of surgeons have made important modifications in the technic. Among these are Lempert, Shambaugh, Meltzer, and others. It must be remembered that the operation is still undergoing changes and that the future will undoubtedly add more. Since some of the changes have resulted in very definite improvements in results, it seems that we can quite confidently expect that future modifications will solve the serious problems still to be faced before the operation can be considered to be perfected. The fenestration operation of Lempert has replaced all earlier types of surgical procedure and is now being performed all over the world. By it, with modifications, the results now being secured by many surgeons surely make clear to any informed if impartial observer that it does indeed mark a very great advance in otology.

It must be admitted that the operation has been much criticized, chiefly for two reasons. The changes in technic as announced from time to time have been understood by some to indicate a continued lack of successful results obtained by previous

*Read at Meeting of the New Orleans Graduate Medical Assembly, March 1948.

technics. We shall see that this is far from the truth. In the second place, it is true that the operation of the present day does not afford satisfactory results in every case. Nevertheless the reports of many surgeons make it evident that fenestration offers the hope to a large proportion of those who are seriously handicapped by deafness due to otosclerosis that the hearing will be restored to a useful level and obviate the need for a hearing aid.

One must remember that until ten short years ago there was no means of restoring the hearing of these individuals in the prime of life other than by the use of a hearing aid. No method of treatment, either medical or surgical, brought about sufficient improvement in the hearing to restore it to a practicable level.

THE DIAGNOSIS OF CLINICAL OTOSCLEROSIS

Clinical otosclerosis is that form of the disease in which progressive deafness of conduction type is produced by the growth of new otosclerotic bone about the footplate of the stapes in the oval window until it is ankylosed. Foci of similar new bone have been shown to exist quite frequently in other areas of the temporal bone by pathologic examination; this incidence may be as high as 20 per cent. These foci produce no known symptoms and their presence cannot be diagnosed.

The chief symptom of ankylosis of the stapes is the gradual onset and slow but sure progression of a typical conduction deafness. With this there may or may not be tinnitus; when this is present it may be mild or quite severe. The deafness is usually bilateral and is very often quite equal in the two ears. It may, however, begin in one ear and exist as a monaural deafness for a considerable time before the other ear is sufficiently involved to show deafness. The process may be much more advanced in one ear than the other, but the type of deafness is always the same in both ears.

Typical conduction deafness has the following well-known characteristics: (a) in the early stages of development of a progressive type, the loss for hearing of low tones by air conduction is greater than that for high tones; (b) hearing by bone conduction is completely normal in the early stages; (c) in later stages, the losses for both low and high tones by air conduction are about equal; (d) hearing by bone conduction shows only mild loss even when hearing by air conduction is quite badly impaired; (e) the Rinne test is consistently negative until the progress of the disease is very far advanced.

Since clinical otosclerosis produces such typical conduction deafness it is evident that the diagnosis is made very largely upon the results of tests of the hearing. These should be made in a quiet room on at least two occasions. The spoken voice at an unaccented conversational level and the forced whispered voice will serve as the first coarse screen to indicate that there is sufficient deafness to warrant further study. The voice tests should be made in each ear separately while the other ear is masked by having the patient rub it with the flat of the hand. Voice tests are only a rough method, but since clinical otosclerosis typically causes losses below the 30 decibel level in the usual speech frequencies, voice tests are well worth while.

The next step in the screening process is careful and repeated testing of air and bone conduction with tuning forks. If the full set of forks from 128 to 2048 double vibrations is available, all should be used. Even if only one or two forks such as those of 512 and 2048 double vibrations are at hand, careful testing will usually give the examiner a very good clue to the presence of a conduction deafness as differentiated from a perception hearing loss.

These tests may seem laborious and time-consuming. There is one method which gives very accurate results and which is quite speedy. It is the performance of the Rinne test by the method of Fowler, Senior. Each fork is given a standard blow; the fork blades are first held one inch from the auditory meatus and the time noted, or a stop watch is started. The patient indicates that he hears the sound by raising a finger. The end of the stem of the fork is then

pressed firmly against the mastoid process over the antrum. The position of the fork is changed from meatus to mastoid and back every four seconds. When the patient indicates that he no longer hears the fork in one position, the elapsed time is noted. The fork is still changed until it is no longer heard in either position and the time is again noted. The duration of the hearing for each fork by both air and bone conduction is thus secured and can be compared, by one test.

In clinical otosclerosis it is characteristic that bone conduction is either quite normal, or that it is better than air conduction, and hence the Rinne test is definitely negative. This indicates a conduction type of deafness; if otoscopy fails to reveal the cause of such deafness by obvious changes in the external or middle ear, it is almost certain that clinical otosclerosis is present. When this is found, it is essential to secure an audiogram. This is of course the modern method of recording the hearing by both bone and air conduction through the normal frequency range, and of determining for certain that a conduction rather than a perception type of deafness is present.

The typical audiogram in early or mild otosclerosis reveals that the low tone loss by air conduction is greater than the high, and that the bone conduction is normal (Figure 1, a). In more advanced cases loss for low and high tones by air conduction is at about the same level; bone conduction is still normal or shows only mild losses

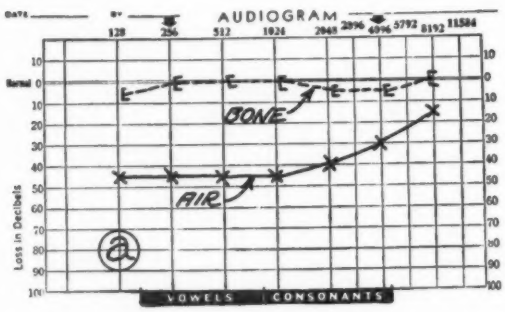
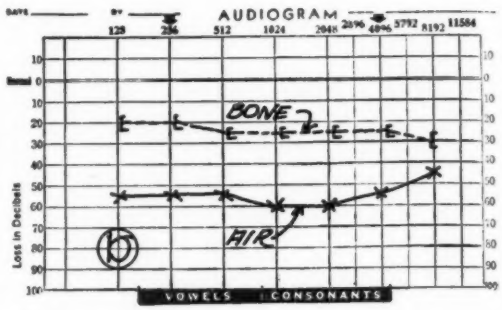


Figure 1. (a) Typical audiogram in early or mild otosclerosis.



(b) Typical audiogram in fairly well advanced otosclerosis.

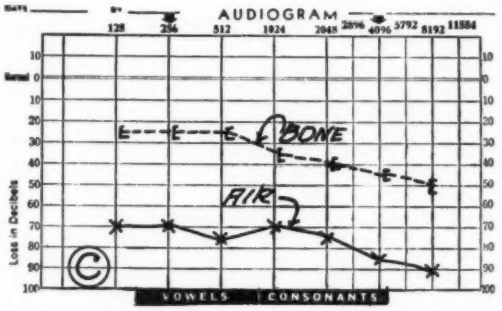
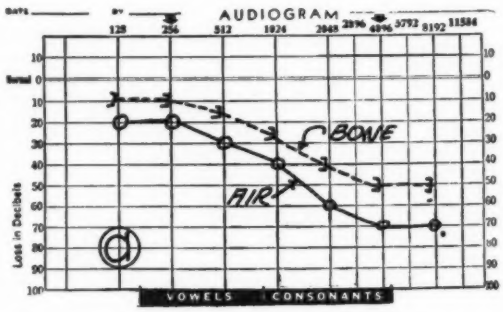


Figure 1. (c) Typical audiogram in far advanced otosclerosis.



(d) Typical audiogram in perception type deafness for which fenestration is absolutely contraindicated.

(Figure 1, b). When the deafness has existed for a long time, or when progress of the loss has been rapid, there will be a considerable loss for low tones, and an increasingly severe loss for high tones by air conduction, with a definite loss of bone conduction, usually most marked for the

higher frequencies (Figure 1, c). This latter type of audiogram is often seen in patients in middle life in whom the deafness has been present for twenty years; it may be noted in quite young patients whose hearing loss has been rapid and who have what is usually termed malignant otosclerosis, with a bad prognosis.

While the diagnosis of clinical otosclerosis thus rests largely upon the hearing tests, especially repeated audiograms, a complete history and physical examination are also necessary. The points in the history suggestive of otosclerosis are: (a) otosclerosis tends to occur in families; (b) it is far more frequent in women; (c) the deafness is usually bilateral and severe enough to handicap the patient before thirty years of age, since it probably begins at puberty; (d) paracusis in which the hearing seems better in very noisy surroundings is usually present; (e) a history of previous serious, prolonged or recurrent middle ear infection is usually not obtained; (f) tinnitus may or may not be present.

In the local physical examination the drums will usually be normal or show only the mild dullness and retraction so often seen without appreciable deafness. They may be very thin and brilliantly shiny. The pink blush of the congested promontory often described is only occasionally seen. Wide external auditory canals with little cerumen and diminished sensitivity are noted but are not characteristic. On inflation of the auditory tube it will be found to be normal. Mastoid radiographs will usually reveal a normal pneumatic structure, although partially or completely undeveloped forms may co-exist with otosclerosis.

INDICATIONS FOR FENESTRATION

Fenestration is indicated only in disabling conduction deafness due to otosclerosis; it is completely useless in perception deafness of any type (Figure 1, d).

It is now generally agreed that unless there is a deafness of 30 or more decibels in each ear through the speech frequency range from 256 to 2048 double vibrations, the disability is not sufficient to warrant the operation. If the deafness is somewhat less than 30 decibels in one ear, it should be below this level in the deafer ear.

Since the object of the operation is to restore the hearing in the operated ear to the serviceable level or better, so that there is less than a 30 decibel loss through the speech frequency range, and since experience has shown that the gain that may usually be expected from the successful operation is of the order of 30 to 40 decibels, it is evident that binaural losses much greater than the 70 decibel level are almost certain to preclude a useful restoration of the hearing. Thus the maximum binaural loss suitable for operation is about 70 decibels through the speech frequency range.

The operation is indicated when the bone conduction hearing is normal or nearly normal through the frequency range from 512 to 2048 double vibrations. It will usually prove of doubtful success when there is any decided loss in bone conduction hearing; this means a loss of more than 30 decibels in one of these frequencies. The operation will usually prove unsuccessful when there is a bone conduction loss of 35 or more decibels in more than one of the frequencies from 512 to 2048.

THE CHOICE OF PATIENTS SUITABLE FOR OPERATION

From the foregoing indications it becomes evident that the patient may be considered as suitable for fenestration only when the requirements outlined are fulfilled. An *ideal case* in which restoration of the hearing to the serviceable level can be expected with very reasonable certainty is one in which bilateral hearing loss by air is 30 or more decibels but not more than 60 decibels, with normal or nearly normal bone conduction hearing, in good general health. The *suitable case* in which a satisfactory result is at least probable is one in which the binaural loss by air conduction is not greater than 70 decibels, and in which the bone conduction loss for the 512 to 2048 frequencies reaches but does not exceed the 30 decibel loss level. The *problem case* in which a favorable result is possible but not too likely to occur is one with

a binaural loss of air hearing greater than 70 decibels, with a loss in bone conduction greater than 30 decibels in one frequency from 512 to 2048 double vibrations.

CONTRAINDICATIONS TO FENESTRATION

As previously stated, fenestration is useless in perception deafness of any type. It is contraindicated in advanced otosclerosis when the air conduction loss is more than the 75 decibels and when the bone conduction loss at more than one frequency between 512 and 2048 double vibrations is greater than 35 decibels; in these patients the so-called cochlear reserve, or actual ability to hear even if the conduction of sounds were restored to normal, is so small that serviceable hearing can scarcely result.

Fenestration is contraindicated in the presence of dermatitis of the pinna or external canal, and in the presence of a chronic drum perforation of any type. Mere scarring of the drum from previous, long-healed otitis media is not a contraindication. The operation must not be done in the presence of active syphilis or tuberculosis, or in diabetes or chronic nephritis for obvious reasons. It should not be done in marked hypertension or with any blood dyscrasia which predisposes to capillary bleeding during or after the operation, since bleeding into the fistula in the labyrinth is almost certain to produce damage to the cochlea which causes further hearing loss.

The operation should be postponed when there is any suspicion of acute upper respiratory infection, especially with the slightest involvement of the auditory tube or middle ear.

PHYSICAL EXAMINATION

It is evident that when a patient is selected for operation on the basis of an accurate diagnosis of otosclerosis and an appraisal of the severity and type of hearing loss, a complete survey of the physical status is required before fenestration is offered. In addition to a careful otoscopy there should be an examination of the entire upper respiratory tract, inflation of the auditory tube, a general physical examination, a complete blood count with determination of the clotting and bleeding time, a blood Wassermann and urinalysis.

In addition it is wise to make an estimation of the psychic status of otherwise suitable patients. Since not everyone will secure a fully satisfactory result even when he meets the physical requirements fully, or after securing a good increase in hearing, loses it again because of closure of the fistula, certain highly neurotic and very unstable types may have to be refused operation. Failure in these rather rare patients will bring about a mental depression of a severe nature. They do best when fitted with a proper hearing aid.

THE RESULTS OF FENESTRATION

Much criticism of the results of the operation has been expressed and sincere doubt brought to the minds of many physicians, both in and out of the field of otolaryngology, by several factors. It is freely admitted by all honest operators that a certain if quite small number of cases in which every known requirement is met and on whom a technically perfect operation has been done without any untoward event in the healing period, do not secure any appreciable improvement in hearing, or the improvement is too small to be of practical importance. These patients are considered to have had serous labyrinthitis due to the entrance of blood and tissue fluids into the fistula, during and after the trauma of creating the window into the labyrinth. Despite many efforts to prevent this occurrence, with considerable success, it cannot always be avoided. It is thus one of the unsolved problems of fenestration.

In a second, much larger group of fully suitable cases operated on with perfect technic in the light of present day information, an excellent early result is followed after weeks or months by a swift or more gradual loss of hearing until it returns to the preoperative level. This is due to regeneration of new bone which completely fills the fistula and again prevents the sound waves from entering the membranous labyrinth. This constitutes the greatest problem of fenestration. Thus far, despite much thought and study and a considerable

amount of animal and human experimentation, no certain means of preventing this bone regeneration has been found. It is the sincere hope of all that continued investigation will finally yield a method of controlling bone regeneration. This will mark a forward step secondary in importance only to the initial development of the operation itself.

It must be pointed out that such bone regeneration does not always occur by any means. It is not the rule and newer technics have steadily lessened its incidence. It does occur with sufficient frequency to be a great disappointment to both patient and surgeon.

Thus far the chief method of procedure when regeneration of bone closes the fistula has been by revision. By a short and simple second operation the fistula area is exposed and a new fenestra is created. This may be done more than once if it is followed by a reasonably long period of satisfactory hearing, in the hope that ultimately bone regeneration will cease. It is not uniformly successful however, as it is followed by useful hearing in only a moderate percentage of patients. It does not supply a satisfactory answer to the problem of fistula closure.

Finally, doubt as to the results actually being secured even by the leading surgeons performing many fenestrations has been created by a most unfortunate tendency for dissention among them over the manner in which results have been assessed, especially in quoting percentages of successful and unsatisfactory cases. There has been a further, if understandable uncertainty created by some leading surgeons who, sometimes on the basis of quite slender evidence, announce some modification in technic which they flatly state to be, or clearly infer, is the final answer to one or other problems of the operation. At a later time they make a further announcement which partly or completely disregards the first, without taking the trouble to explain that the previous technic proved unsatisfactory for one reason or another. This is most unfortunate since it is unnecessary and creates confusion and doubt.

It is hoped that the really significant and scientific research which is now being carried on in several centers will yield some of the answers to the problems, and that the results will be announced to the waiting world only after confirmation and proper statistical study, in a manner which will restore the confidence of all in this quite wonderful advance in otology.

CONCLUSIONS

Despite serious problems still awaiting full solution, despite criticism and disbelief, in the face of the technical difficulties of the operation, which is surely one of the most intricate and delicate in the whole realm of surgery, and notwithstanding the very great disappointment to patient and surgeon when it fails for any reason, there can be no doubt but that fenestration for clinical otosclerosis is here to stay. Even with its uncertainties and present day imperfections, it can lead to the rehabilitation of persons seriously handicapped by deafness in the very middle of the best years of their lives. It can restore them to happy human beings, socially and economically. It does this, make no mistake, even if not as often as anyone could wish.

The future is certain to bring forth improvements in technic by which the operation will be perfected. This is not too much to expect in the light of the fact that the modern operation of fenestration is now only eleven years old.

While we all wait for this future to become the present, it is the humble opinion of the author, based on his own experience, that all otolaryngologists should put aside their doubts and skepticism regarding the fenestration operation. He suggests that they study the indications for the operation closely, be on the watch for and select suitable cases carefully, and offer them the operation at the hands of a fully trained and highly skilled surgeon. This should only be done after a most careful and honest explanation to the patient of the possibilities for both success and failure, leaving the choice completely in the hands of the

patient. He feels strongly that this constructive and optimistic though realistic course will even now be followed by success often enough to make it clearly the plan of choice in the rehabilitation of disabling deafness due to clinical otosclerosis.

REFERENCES

Lempert, Julius: Arch. Otolaryngol., 27:555, 1938.

Shambaugh, G. H., Jr.: New Orleans M. and S. J., 94:105, 1941.

REMOVAL OF THE AURICULAR APPENDAGE AND ITS POSSIBLE THERAPEUTIC USE

A PRELIMINARY REPORT

WALTER J. BURDETTE, Ph. D., M. D.*

NEW ORLEANS

The treatment of auricular fibrillation is not altogether satisfactory. It is not possible in all cases to effect a conversion to normal rhythm by using quinidine and digitalis, and even if normal rhythm is resumed, emboli already formed may be dislodged by contraction of the auricle. Anticoagulants are useful when emboli complicate the arrhythmia, but it is not feasible to continue their use indefinitely and of course clots already present are a constant source of danger. It seems reasonable that removal of that portion of the auricle where the situation is most favorable for clotting during fibrillation may be useful in treating patients who do not respond to medical therapy. By removing the auricular appendage the channel for circulation of the blood is decreased in size, and the reservoir for thrombi is attenuated. The result should be a diminution in complications due to emboli and an increase in the aid which can be offered to the patient afflicted with auricular fibrillation.

A suitable technic for removing the auricular appendage is desirable and has been worked out in the laboratory during the past two years. Twenty dogs were used and both auricular appendages were removed in each. In 16 the operation was done in two stages, and in 4 it was done in one stage. All animals survived removal of either the right or the left appendage. When the remaining appendage was removed as a second stage, all lived except one animal which died from an overdose of nembutal in the immediate postoperative period. Three of 4 dogs died after both appendages were removed in one stage. Autopsy revealed pneumonia of the left lower lobe in 1 dog and atelectasis in all 3.

Observation of the living animals for a period up to two years showed no detectable deleterious effect of the operation. They gained weight which was not due to edema, proved at autopsy, and one delivered a litter in the postoperative period.

The technic of auricular appendectomy was evolved with the objectives of safety and simplicity in mind. The animals were anesthetized with intravenous nembutal, and positive intratracheal pressure was supplied during operation. The chest was entered through the third interspace. Because of the rotation of the heart, the pericardium was opened anterior to the phrenic nerve on the right and posterior to the nerve on the left. The tip of the appendage was grasped with a hemostat, and the walls of the base of the appendage, including a portion of adjacent auricle, were sutured together with interrupted silk, mattress sutures. Adjacent sutures were then tied to each other. A linear closure resulted. Before removing the isolated appendage, a small opening in the distal portion proximal to the hemostat was made in order to check the presence of a competent suture line. The pericardium and chest wall were closed with interrupted sutures. The operation visualized in man would include opening the tip of the appendage carefully for removal of any clot present, the application of a hemostat, and the procedure described above.

At autopsy the auricular stump was well healed in all cases, and microscopic sections showed endothelium covering the scar. There were no signs of cardiac failure or embolic phenomena. The average weight of the right auricular appendage was 756 mg.,

*Department of Surgery, Louisiana State University School of Medicine, New Orleans, La.

of the left was 704 mg. The average weight of the heart was 109.8 gm.

The electrocardiogram showed some minor changes in the P wave. The V leads were taken with the electrode at the level of the auricle. Removal of either right or left appendage or both resulted in decreased height of the P wave in leads I, V_2, V_4, and V_5. There was diminished negative deflection of the P wave in leads V_1 and V_2 after removal of both or either appendage.

Results of these experiments indicate that removing one auricular appendage is not likely to be attended by excessive mortality. This operation should lower the mortality from emboli arising in a fibrillating auricle. An additional possibility is the effect of such a procedure on the fibrillation itself. The circus movement which is present in fibrillation may be stopped by shortening the pathway, speeding conduction, or lengthening the refractory period. It is conceivable that shortening the pathway surgically, alone or in conjunction with drugs, might alter or even convert the arrhythmia. This was not found amenable to experimental testing. There seems, however, sufficient justification otherwise for the procedure in man and the answer as to whether fibrillation itself will be altered can be determined in that manner. There is no desire to alter or detract from successful medical management of auricular fibrillation, but it is believed that in a selected group of cases the proposed operation may offer the patient some additional promise.

—o—

LOUISIANA STUDY OF CHILD HEALTH SERVICES[16]

CHAPTER IV

PRIVATE PRACTICE

PHYSICIANS

As of May 1946, there were 1487 physicians engaged in private practice in Louisiana; there were 522 children for every physician. The national average was 310 children per physician. Among the forty-eight states, this figure varied from 143 to 764; Louisiana ranked thirty-seventh among all states in this respect. In the southeastern region of the country, with an average of 588 children per physician, Louisiana ranked high. Figure 6 indicates Louisiana's position relative to other states and regions. Because this was a study of child health services, number of children, rather than total population per physician, has been used to indicate relative accessibility of physicians to children. There were 42 women physicians in Louisiana and 11 of them were pediatricians. All but 58 of 956 general practitioners and all but 1 of 57 pediatricians were white. Forty-six per cent of the state's doctors were under 45 years of age, 38 per cent were between 45 and 65 years of age, and the remaining 16 per cent were older.

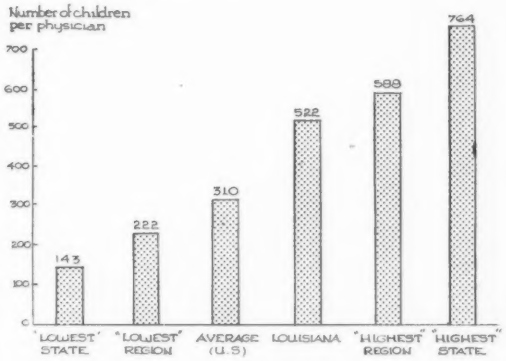


Figure 6. Children per physician; Louisiana compared with other states and regions of the United States.

It is realized, but must be emphasized again, that the number of *all* doctors—general practitioners, medical specialists, and dentists—in Louisiana is greater now (October 1948) than it was when this study was made.[17] There is no doubt that improvements in distribution have taken place as well.

Using the same type of classification for

[16]This is the second of a series of three reports on Health Services for Children in Louisiana, presented by the Louisiana State Pediatric Society and the American Academy of Pediatrics; the first appeared in the October issue of this journal.

[17]Louisiana supplied far more than its quota of doctors for the armed forces and in May of 1946, many physicians had not yet returned to practice.

parish or county types as was employed in the national study, and also in the first three chapters of this report, again it appears from the data in table V that children who live outside of our metropolitan parishes had relatively little available medical care.

TABLE V

NUMBER OF PHYSICIANS AND NUMBER OF CHILDREN PER PHYSICIAN, BY PARISH TYPE*

Parish Type	Number of Physicians	Number of Children per Physician
Lesser metropolitan	832	255
Adjacent	87	1131
Isolated semirural	471	787
Isolated rural	97	980
Entire state	1487 (total)	522 (average)

*As of May, 1946.

PEDIATRICIANS

Fifty-seven physicians in Louisiana reported that they limited their practice to children—for each of these there were 13,612 children. Among other states,[18] this figure varied from 4,182 to 73,005, and the average for the nation was 10,299. Of the 57 pediatricians in Louisiana, 16 (35 per cent) had been certified by the American Board of Pediatrics; over the entire country, 51.4 per cent of these physicians were so certified. All of the pediatricians in Louisiana were located in centers having populations of 10,000 or more; 48 of them were in the three metropolitan areas and the other 9 were practicing in isolated semirural parishes.

OTHER SPECIALISTS

There were 474 specialists other than pediatricians; their distribution by specialty and certification appears in Table VI.

TABLE VI

NUMBER OF SPECIALISTS, AND NUMBER CERTIFIED, BY SPECIALTY*

Specialty	Total Number	Number Certified by Specialty Board
Internal Medicine	111	38
Allergy	6	1
Psychiatry and Neurology	17	7
Surgery, except Orthopedic	123	38
Orthopedic Surgery	16	8
Obstetrics and Gynecology	54	16
Ophthalmology and Otolaryngology	119	50
Radiology, Anesthesiology	28	18

*As of May, 1946.

TRAINING OF PHYSICIANS

Five hundred and two of the 956 general practitioners gave information as to their hospital training; 13.8 per cent reported that they had received none at all after graduation from medical school, and an additional 2.5 per cent stated that they had had less than a year of such training. About 41 per cent of the general practitioners reporting had received between one and two years of hospital training, and 43 per cent of them had had two or more years. One can conclude, therefore, that at least one out of six physicians in Louisiana had received little or no hospital training; this same deficiency was found for about one out of five physicians over the United States generally.

More specifically for the purposes of this report, it was interesting to note that two out of every five practitioners in Louisiana had received none, or less than a month of hospital training in pediatrics. Similar deficiencies were noted for about half of the general practitioners in the entire nation.

PHYSICIANS' SERVICES

Data compiled from one-day reports of practicing physicians appear in Table VII, and permit a number of comparisons as to the total volume of care extended to children in the various parish groups in Louisiana with that found to be average for the United States as a whole, as well as for the highest and lowest ranking states in this regard.

TABLE VII

PRIVATE MEDICAL SERVICES FOR CHILDREN ON AN AVERAGE DAY; COMPARISONS BY REGIONS

Region	Physicians Visits per Day, per 1000 Children (Office, Home, and Hospital): Total	Sick	Well
United States	13.5	9.6	3.9
Louisiana	10.2	8.0	2.2
Metropolitan parishes	15.1	11.3	3.8
Adjacent parishes	6.9	6.0	0.9
Isolated semirural parishes	9.1	7.1	2.0
Isolated rural parishes	6.7	5.9	0.8
Highest ranking state	21.8	14.9	6.9
Lowest ranking state	7.8	6.3	1.5

[18]This and subsequent references to data for other states are taken from the National Report (see previous references).

Again, it appears obvious that children residing in rural parishes receive considerably less medical care in sickness or in health than do those in metropolitan areas. These differences seem to be more reasonably explained by inequitable distribution of physicians than by significantly better health—and consequent less need for medical care—among children living in rural parishes. Apparently, children in isolated rural parishes received less than a fourth as much medical attention when they were well, and less than half as much when they were sick, as did their city cousins.

Seventy per cent of the medical care extended to children in Louisiana was furnished by general practitioners; in the metropolitan parishes, this proportion decreased because of the relatively greater contribution from pediatricians and other specialists. Data of this sort are considered by parish types in the accompanying tabulation.

TABLE VIII

PROPORTION OF MEDICAL CARE RENDERED TO CHILDREN BY GENERAL PRACTITIONERS AND SPECIALISTS, BY PARISH TYPE IN LOUISIANA

	General Practitioners	Pediatricians	Other Specialists
Whole State	70.2%	14.7%	15.1%
Metropolitan	41.1	30.0	28.9
Adjacent	99.1	.....	0.9
Isolated semirural	86.6	5.8	7.6
Isolated rural	100.0	.....	.....

Over the country generally, 75 per cent of children were cared for by general practitioners, 11 per cent by pediatricians, and 14 per cent by other specialists.

NUMBER OF VISITS PER DAY

General practitioners reported seeing an average of 6 children and 14 adults as private patients daily; nationally, the average daily case load was found to be 16 patients per physician. As noted in the table which follows, nearly half of the general practitioners in Louisiana reported that they saw 20 or more patients daily, and 6 per cent of them saw 50 or more patients each day.

TABLE IX

CASE LOAD OF GENERAL PRACTITIONERS IN LOUISIANA ON AN AVERAGE DAY*

No. of visits in one day	Per Cent of Physicians Reporting Specified Number of Visits to: Persons of all ages	Children under age 15
None	18	25
1-9	16	51
10-19	20	19
20-29	20	5
30-39	11	
40-49	9	
50 and over	6	
Total	100%	100%

*See text, under "place of visits", below.

The case load for pediatricians, as determined from records kept for twenty-eight consecutive days for each one, was an average of 21 children daily; the average for pediatricians over the entire United States was 16: pediatricians in Louisiana thus had a case load about one-third heavier than that of their colleagues in most of the other states.

PLACE OF VISITS

Every effort was made to distribute all of the questionnaires from which data as to case loads were derived in such a manner that it would be possible to obtain a fairly representative cross-section, including Sundays, holidays, and days off, in compiling the averages. This explains, of course, why some doctors—whose questionnaire happened to call for work done on a Sunday or during a vacation—reported that they saw *no* patients. From these same questionnaires, information was secured relative to the proportion of visits made to offices, homes, or hospitals; these data appear in Table X.

TABLE X

PLACE OF VISITS TO CHILD PATIENTS: PERCENTAGE BY LOCATION

	Per Cent of Visits Made by: General Practitioners	Pediatricians	Other Specialists
Office	69	73	74
Home	17	13	5
Hospital	14	14	21
Total	100	100	100

SUMMARY

1. Louisiana had 32 per cent more children per physician than the average for the United States generally. Only 11 states had

a higher ratio of children to physicians, but Louisiana ranked slightly higher than the average for 12 southeastern states in this respect.

2. Outside of the metropolitan and adjacent areas of Louisiana, there were only 9 pediatricians, working among 60 per cent of the state's children who resided in 46 of its 64 parishes.

3. Specialized pediatric care was not easily accessible to children in many parishes, particularly those in the central part of the state.

4. Many physicians in Louisiana had had little or no formal hospital training of any sort after graduation from medical school, and about 2 out of 5 of them received none, or less than a month of hospital training in pediatrics.

5. Physicians' services received by children in our isolated rural parishes amounted to less than half of such services received by children in metropolitan parishes.

6. Seventy per cent of the medical care extended to children in Louisiana was furnished by general practitioners.

7. The case load for both general practitioners and pediatricians was about one-third higher in Louisiana than the average for the nation.

DENTISTS

NUMBER, TYPE AND TRAINING

As of June 1946, there were 681 dentists in private practice in Louisiana; 70 per cent of them answered questionnaires from which the data of this section were derived. About 30 per cent of these reported their ages to be less than 45 years, and 7 per cent of them were over age 65. Sixty per cent of the dentists were located in cities with 10,000 or more population, and slightly more than 6 per cent of them practiced in isolated rural parishes. There were 5 or more dentists in 31 parishes, and none in 4 parishes; 9 parishes had one dentist each and the others had from 2 to 5. The number of children per dentist in each of the parish groups appears in Table XI.

TABLE XI
NUMBER OF CHILDREN PER DENTIST, BY PARISH TYPES

Parish Type	Children per Dentist
Metropolitan	604
Adjacent	2342
Isolated semirural	1528
Isolated rural	2105

Thirty-four dentists reported that they limited their practice to one specialty—2 to pedodontia, 14 orthodontia, 3 to periodontia, 9 to prosthodontia, and 6 to oral surgery; all but 4 of these specialists were in the metropolitan areas. Three of the orthodontists and 2 of the periodontists were certified by American Specialty Boards. Two hundred and nine of 221 dental general practitioners reporting on the item stated that they had received no postgraduate training in pedodontia, and only 4 of this number reported that they had received a month or more of such training. Six dentists reported that they were assisted by dental hygienists.

For the entire state, there was 1 dentist for 1,139 children; over the United States generally, there was 1 dentist for 548 children. Louisiana ranks in thirty-fifth place among the states, which vary in this regard from one to 273 up to one to 2155 children. Among the southeastern states, the average was 1428 children for every dentist.

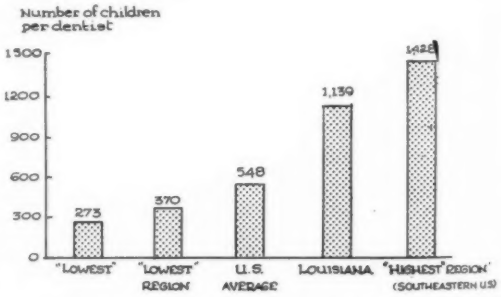


Figure 7. Children per dentist; Louisiana compared with other states and regions of the United States.

DENTISTS' SERVICES

Some comparative data referring to dental care on an average day appeared in Chapter II;[19] Louisiana ranked thirty-fifth among the 48 states. Table XII permits

[19] New Orleans M. & S. 101:176 (Oct.) 1948.

comparisons of the volume of dental care for children furnished in the various parish or county groups of Louisiana and the United States generally.

TABLE XII

DENTAL VISITS OF CHILDREN ON AN AVERAGE DAY, BY PARISH OR COUNTY TYPE

Parish Type	Visits per 1000 Children	
	Louisiana	United States
Metropolitan	2.8	3.5
Adjacent	1.2	2.2
Isolated semirural	1.4	2.0
Isolated rural	1.0	1.1

About 24.8 per cent of the total number of dental services reported were extended to children under age 15; 7.1 per cent to those under age 6, and 17.7 per cent to those between 6 and 15 years of age. The data of Table XIII are based on returns from only 413 dentists; though incomplete, they indicate the type and distribution of dental services rendered to preschool and school age children. These dentists saw 8.2 patients on an average day. There were inadequate data on which to estimate differences in the amount of dental services furnished to white and negro children. Among other southern states, however, ratios for white children per white dentist were from 2.8 to 13 times larger than were those for non-white children per non-white dentist.

TABLE XIII

DENTAL SERVICES FOR CHILDREN ON AN AVERAGE DAY

	Age of Children	
	Under 6 years	Six to 15 years
Extractions	80	109
Fillings	94	324
Other dentistry	28	68
Examinations only	18	154
Total number of children seen	220	655

Of 271 dentists reporting on the number of hours spent in various dental activities during one single month, it was noted that 7 of them contributed an average of three hours each per week to preschool and school dental services, and 19 of them spent an average of five hours each per week in other activities such as teaching, conducting out-patient clinics, or carrying out dental programs in various institutions.

SUMMARY

1. Louisiana ranked low among the states in number of dentists relative to its population of children; there were over three times as many children per dentist in isolated rural parishes as there were in metropolitan areas.

2. Sixty per cent of the state's dentists were located in cities of more than 10,000 population.

3. Very few dentists had any formal postgraduate training in the care of children's teeth.

4. Dental services for children constituted about a quarter of the total volume of care rendered by the dentists of this state.

CHAPTER V
COMMUNITY HEALTH SERVICES IN LOUISIANA

INTRODUCTION

Data have been collected bearing on some, but not all, of the kinds of health services for children usually provided by American communities. Discussion of these services will be largely quantitative, though it must be emphasized that quality of service is also of paramount importance. A child health conference, for example, cannot achieve real health supervision unless the workers —physicians, nurses, nutritionists and others—are well trained, interested, industrious, and really trying to do a good job.

Proper evaluation of qualitative as well as quantitative aspects of all its health services is a continuing responsibility of each local community.

Although, as has been shown, a relatively small proportion of total medical services for children is supplied by community health agencies, they lend themselves so readily to detailed analysis, and throw such light on the whole medical program, that they have been given extensive discussion.

COMPLETENESS OF HEALTH SUPERVISION

Medical well-child supervision merits first attention since it is the keystone of preventive medicine for children. One should know two facts to evaluate need for organized child health conferences: (1) What proportion of children should be registered in these conferences, and (2) how much service is necessary to give adequate care to this proportion. The answer to the

former question depends on a knowledge of the completeness of child health supervision, from all sources. A previous section of this report (Chap. III) has indicated that in Louisiana about 90 per cent of health supervision was given by private physicians and 10 per cent through organized child health conferences. From this ratio and the knowledge that 10,674 different children[20] were reported under care at Child Health Conferences one can assume that about 106,000 children, one third[21] of the children under age 5, were under medical supervision from all sources.

An even more significant measure of completeness of health supervision may be made by estimating the proportion of infants under age 1 who received care. This is the group, above all, for whom medical supervision is most needed. Although, again, the data are not available directly, more than one-third of the babies born in the state that year may be presumed not to have had medical well-child supervision from any source.[22] It is obviously of paramount importance, for further improvement in infant health and continued lowering of infant mortality, to extend health supervision to this group through expansion of services rendered both by practicing physicians and child health conferences.

Just what proportion should be provided by child health conferences depends on a variety of factors, including economic status and availability of physicians. It seems obvious that the 10 per cent provided in Louisiana was inadequate for the state as a whole but need will vary from a smaller percentage in relatively well-to-do areas to the great majority in poorer sections. With the goal of completeness ever in mind, plans must be worked out for each community on the basis of local conditions.

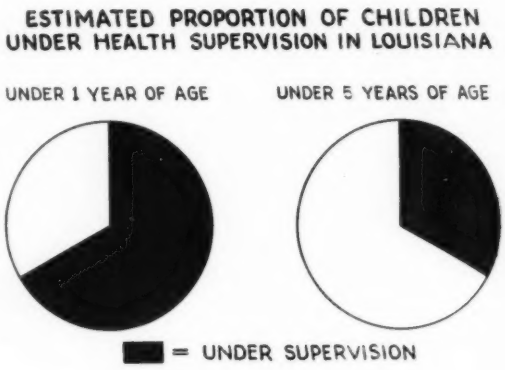

ESTIMATED PROPORTION OF CHILDREN UNDER HEALTH SUPERVISION IN LOUISIANA
UNDER 1 YEAR OF AGE
UNDER 5 YEARS OF AGE
= UNDER SUPERVISION

Fig. 8

COVERAGE BY CHILD HEALTH CONFERENCES

Variation in numbers of children under supervision within Louisiana and in the nation as a whole is presented in Table XIV.

[20]This figure is actually high, since it is customary in clinic records to count infants and preschool children separately. Thus, when a child passes his first birthday during the year he is counted twice in this total.

[21]This proportion may also be estimated in another way. It has been noted earlier (Chap. III) that on a single day 3.2 visits per 1,000 children under age 5 were made for health supervision. If this rate continues daily throughout the year, and visits per child to physicians and conferences are at essentially the same rate, a total of 1160 visits per 1,000 children would be expected in a year.

Most pediatricians aim at monthly visits during the first year, four to six visits during the second year and visits two or three times a year thereafter. Clinics, in view of limited facilities, use a lower standard of nine visits in the first year and two a year thereafter. Since 1,000 children under 5 will include approximately 200 in each year of age, a very conservative estimate of visits needed would be 200 x 9 equals 1800; plus 800 x 2 equals 1600; or a total of 3400, almost three times as many visits as were actually made that year.

From these approximations, it is probably fair to say that two-thirds of the children in Louisiana under 5 years of age were not receiving medical well-child supervision at the time of this study.

[22]The proportion of children registered in Child Health Conferences who were under age 1, was reported for 40 per cent of the State total. If this proportion holds for the entire group, 5,500 of the 10,674 children registered were under age 1. In order to relate this to the births which took place during the year it is necessary to apply a correction factor (Wegman M. E. and Bellows, M. T.: Indices and Standards for Child Health Services, American Journal of Public Health, 35:715, July, 1945.) which may be estimated as two-thirds, since the actual correction factor cannot be calculated from the data available. The number of infants coming under care for the first time in Child Health Conferences would therefore be 3,665. Using the 90-10 ratio referred to earlier, one many conclude that, in all, 37,000 infants came under medical well-child supervision during the year. This constitutes 64 per cent of the 57,838 births which took place in the year 1945.

Metropolitan areas in the state had better than double the state average; on the other hand, isolated rural areas, containing 12.2 per cent of the children under 5, had only one third the state average.

The Louisiana average, 33 per 1,000 children under 5, was about half the national average; 33 states reported higher figures. In eight states, moreover, the average was well over 100, and in one it was 265, indicating that there were eight times as many children under health conference supervision, proportionally, as in Louisiana. The discrepancy was greater in rural areas; Louisiana figures for metropolitan and adjacent areas are almost up to the national average for this segment of the population.

TABLE XIV

CERTAIN CHARACTERISTICS OF CHILD HEALTH CONFERENCES, BY PARISH (OR COUNTY) GROUPS

	Children Registered	Visits	Sessions	Attendance per Session	Visits per Patient per Year
	per year, per 1000 population under 5				
U.S.A.—total	62.3	181.5	11.4	15.9	2.9
Lesser Metropolitan	79.7	220.7	14.1	15.7	2.8
Louisiana-total	33.0	109.5	7.8	14.1	3.3
Lesser Metropolitan	74.9	319.7	15.8	20.2	4.3
Adjacent	24.1	34.9	5.7	6.2	1.4
Isolated Semirural	15.4	23.3	4.4	5.3	1.5
Isolated Rural	10.5	22.2	3.6	6.1	2.1

NUMBER OF CONFERENCE SESSIONS

To provide health supervision through child health conferences 2509 sessions were held in the year of the Study—7.8 per 1,000 children under 5, 68 per cent of the national average (Table XIV). If these conferences gave no greater proportion of child health supervision than was reported, it can be estimated that four times as many clinic sessions would be needed to discharge the responsibility adequately.[23]

[23] If as many as 12 children can be seen effectively in a two hour session (an average of only ten minutes per child), at least 28 sessions per 1,000 children under 5 would be necessary. (3400 visits needed for 1,000 children; 10 per cent to be provided by child health conferences equals 340; divided by 12 patients per session equals 28.)

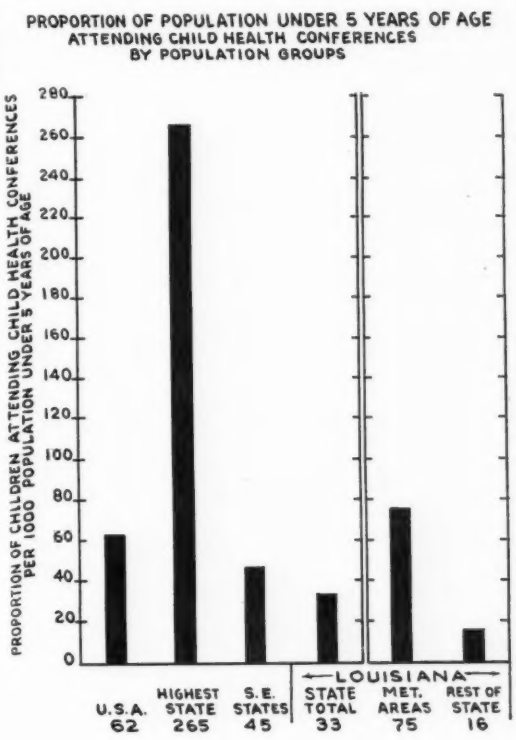


Fig. 9

Figures for the state as a whole are to some extent misleading because of variations by parish groups. In metropolitan areas, for instance, the number of sessions available was twice the state average and better than the national average for areas of this kind, despite the fact that Baton Rouge, capital of the state, had no child health conferences at all. In isolated rural parishes, by contrast, the sessions were about one-eighth of calculated need. In 31 of the 64 parishes, furthermore, there were no conference sessions at all; 34 per cent of the state's newborn children in 1944 lived in these parishes. For white births this figure was 32 per cent while for colored births it was 38 per cent, i. e., almost two-fifths of the colored child population had no public health conference readily available. In the nation as a whole, 31 per cent of the children under 5 lived in counties which had no child health conferences.

As expected, one finds the lowest amount of clinic service available in that very area

which has the lowest amount of service by private physicians. It is these same isolated rural areas which generally have the highest infant mortality rates.

ATTENDANCE PER CONFERENCE SESSION

In any conference session, considerations of economy and efficiency compete with the need for spending an adequate amount of time with each child. The average conference calls for two hours of the doctor's time. Desire for economy often results in aiming at an average of 20 patients, which allows him the grossly inadequate time of six minutes per child. Furthermore, variations in attendance, because of weather and other conditions, mean some sessions may have as many as 30 patients. This puts a frustrating burden on the physician, and also provides an almost insignificant amount of time for each child. Poor attendance at other sessions lowers the average but is no compensation.

In Louisiana, urban conferences showed overcrowding not conducive to the best work. The average number of patients at a session exceeded the national average for areas of this size by almost one-third. In rural areas the average attendance was much lower but one must not conclude that the average time spent with each child was necessarily greater. So many demands are made on the Health Officers in rural Louisiana that child health conferences are frequently part of the so-called "general conference". During the same two hour period, not only children are seen, but also patients with tuberculosis, venereal disease or other conditions. The "general" clinic is not a desirable arrangement and has many dangers, but inadequacies of staff often dictate such compromises with standards. As mentioned earlier, the very nature of rural geography and distribution of population makes it inevitable that average attendance will be lower than in cities.

AMOUNT OF SERVICE PER CHILD

As pointed out earlier, health supervision, to be meaningful, needs to take place at regular intervals, spaced properly to follow growth and development closely. It must be reemphasized that number of visits per child is only part of the story. Quality of service and time spent at each visit are major determinants of success.

Infants should be seen more frequently than preschool children but the data give only combined averages. An average for our state of 3.3 visits per year, per child registered (Table XIV), is higher than the national average and, as a matter of fact, even approaches the nationwide average for "greater metropolitan areas". This is due chiefly to the superior record in the "lesser metropolitan areas" of the state, whose record exceeds the national average for such areas by over 50 per cent.

The average number of visits per patient is probably influenced chiefly by two factors: (1) quality of service as an inducement to patients to return; and (2) frequency with which clinics are held. In a city, clinics are held daily or several times a week in one location; whereas in an isolated rural area there may be too few patients to justify a clinic session more than once a month. If some untoward incident keeps a child away on a given day, a whole month or longer must elapse before he may attend another clinic session.

One can, to a very limited extent, evaluate completeness of service to children registered in child health conferences by comparing the total number of visits which should have been made with those actually recorded. Such an analysis indicates a statewide performance of over 80 per cent of that expected by minimum standards. More satisfying is the record of official agencies in the state's metropolitan areas where performance matched expectation almost exactly.[24]

[24]Using the minimum standards cited above (Footnote 21) the 3665 different infants estimated to have been under supervision (Footnote 22) should have made 9 visits each, and the 5200 preschool children 2 visits each, a total of 43,000, compared with 35,000 visits actually recorded. For the official agencies in metropolitan areas, moreover, expected performance, making the above assumptions, is 26,200 visits and actual performance is 26,548, indicating very creditable work, even though short of the optimum.

STAFF

Contrast between rural and urban areas in regard to staff in clinics is very striking. In metropolitan areas of the state the great majority of sessions, 82 per cent of the total, were conducted by pediatricians. In the rest of the state, no pediatricians at all were in charge. Responsibilities here were divided between general practitioners and health officers who "doubled" as clinic physicians, in the proportion of 54 to 46, similar to the national average.

Consultation services to clinics by nutritionists were reported as good but services by psychiatrists and psychologists were almost nonexistent. In metropolitan areas, however, such consultation services were reported as being available in more than 80 per cent of clinic sessions. "Available" probably means in the community rather than in the clinic itself.

PUBLIC HEALTH NURSING SERVICES

Directly related to child health conferences are public health nursing services. It is almost axiomatic that a child health conference is not likely to be effective without public health nursing follow-up, yet 24 per cent of conferences in the New Orleans area, reported as not under health department auspices, were without this service. At the time of the survey, Louisiana had a total of 195 full-time public health nurses, 1 per 12,000 population or 25 per 100,000 children (Table XV); 10 parishes (16 per cent) had no public health nursing service at all.[25] This deficiency in number of public health nurses is one of the most important limiting factors in extension of child health services.

TABLE XV

PUBLIC HEALTH NURSING SERVICES BY PARISH (OR COUNTY) GROUPS

	Public Health Nurses per 100,000 children		Home Nursing Visits per 100,000 children	
	U.S.	La.	U.S.	La.
Total	40	25	210	75
Lesser Metropolitan	55	44	303	149
Adjacent	27	13	125	41
Isolated Semirural	24	19	94	51
Isolated Rural	15	20	48	40

The National Organization for Public Health Nursing has set as a minimum standard 1 nurse per 5,000 population and strongly recommends 1 to 2500, or 125 nurses per 100,000 children. Louisiana would need five times as many nurses as are now employed, in order to reach this standard. The best state in regard to public health nursing service reported an average of 101.1, four times Louisiana's average, and 34 states reported figures higher than Louisiana's. Even our metropolitan parishes reported figures well under the national average for areas of this type, and other areas of the state had less than half the metropolitan average.

The educational preparation of public health nurses in Louisiana was not adequate. Of the 194 reporting on preparation, only 47 (24 per cent) had completed a full year of public health nursing training; the rest had none or less than one year. The duties of the public health nurse are different from those of the well trained hospital nurse and public health training makes up little of the undergraduate curriculum in schools of nursing. Since a considerable amount of teaching is an integral part of a public health nurse's daily work it is essential that she receive special training in this field.

Rates for home nursing visits to children appear to be related at least in part to distance, since this figure is higher in more densely populated states. Thirty seven states have figures higher than those in Louisiana, and the average in the highest state, 858, was more than ten times Louisiana's. Again the metropolitan areas stood out in comparison with the rest of the state.

Only one parish in the state, Orleans, had service which could be classed as complete with regard to all four essentials for public health nursing service to children (Child Health Conferences, home visits for health supervision, school nursing, bedside nursing). Three states indicated that 100 per cent of their counties offered such complete service but the national average was 18.6

[25] In contrast to the increase in number of physicians in practice since the time of the Study, the actual number of public health nurses in the state has decreased more than 15 per cent.

per cent. Throughout the nation metropolitan areas were superior in this category.

If one disregards bedside nursing service, which is often considered separate from general public health nursing, 42 parishes, 66 per cent, had services complete in the other three essentials. The nationwide average was 41 per cent; 5 states reported that 100 per cent of their counties offered such service.

SCHOOL HEALTH SERVICES

Since surveys from various areas have shown that health services available to school children are usually sadly lacking both quantitatively and qualitatively, the mere presence of this service should not be accepted as adequate. Furthermore, 14 parishes, in which 13 per cent of the children in the state resided, had no organized school medical service in public elementary schools, and 9 parishes, with 5 per cent of the children, had no medical or nursing service at all. Metropolitan areas apparently had relatively complete coverage.

Louisiana did better than the United States average in percentage of children living in parishes with school medical service, the latter showing 22 per cent with no service. Statewide practice in Louisiana (except in Orleans parish) of centering medical service for school children in parish health departments, rather than setting up separate departments of health service in the school system, is probably responsible for this superiority.

Only 2 pediatricians actually worked in schools and both of these were in metropolitan areas. In the whole state, other than the 30 Health Officers (who covered 55 parishes), most of whom did some part-time work in the schools, there were only 7 physicians, including the 2 pediatricians mentioned above, giving services in the schools. Twenty-one nurses in New Orleans and 1 in another parish did school work exclusively. Elsewhere in the state, service was given under the more desirable generalized public health nursing program.

Analysis of school services available by race shows that colored children were somewhat worse off than white. Seventeen per cent of the colored and 13 per cent of the white school children lived in parishes that had no school medical services. There were no differences in metropolitan and adjacent areas, although, as pointed out above, we have no information as to relative adequacy of the service in the various schools. In isolated rural areas 22 per cent of colored and 12 per cent of white children lived in parishes that had no school medical services.

DENTAL CARE

Deficiencies in dental care are among the greatest public health problems facing our country. Louisiana is no exception. It is extremely difficult to set proper standards for amount of dental clinic care necessary, but the inadequacy of the present situation speaks for itself. A total of 2,966 hours of dentist service was given by official agencies, and 1,408 by voluntary groups. For the population in the state under 15 years, this is an average of six hours per 1,000 children per year (less than one half a minute per child), compared to a national average of twenty-seven hours per 1,000 children. The highest state in this regard had a figure of 121 hours per 1,000 children or twenty times the service in Louisiana. Thirty-three states ranked higher than Louisiana in this cateogry.

It is of interest to note that 32 per cent of the total dental clinic services was provided by voluntary agencies; in medical services the figure was 13 per cent. Distribution of dental clinic service was even more disproportionate than medical. Six parishes of the 64 in the state had dental clinics and in 2 of these voluntary groups were working.

SPECIAL SERVICES

One comment that can be made on the special services analyzed is that they were few and far between. Mental hygiene services were available only in New Orleans. Orthopedic clinics were better distributed, being held in eight centers with attendance by patients from all over the state. There were no centers at all reported for rheumatic fever, vision, or hearing,

but there were two reported for speech training.

No mental hygiene service was given by official agencies and only one voluntary agency was active, in the New Orleans area. Here, too, on a quantitative basis, the number of children cared for is very small, totalling only 217 for the whole community, obviously inadequate. This is borne out by a comparison with a national average of 2.2 mental hygiene clinic patients per 1,000 children, with Louisiana near the bottom of the list with a figure of 0.3. Sixteen states had lower figures, including 12 states with no such service at all.

The total number of physically handicapped children cared for was 2,320, an average of 3.0 per 1,000 children. This compares with a national figure of 4.6, but again there is a wide discrepancy. Six states reported an average of more than 10 and one had 14.4. Since it is not likely that Louisiana has fewer physically handicapped children than other areas it appears that a great proportion of these children are not receiving clinic services.

SUMMARY

1. It is estimated that of the children in Louisiana one-third of those under one year of age, and two-thirds of those under five, were not under child health supervision from any source.

2. Three per cent of the state's children under five received supervision through child health conferences—half the national average and three-quarters the average for the southeastern states. Five times as many children, proportionally, were under health conference supervision in metropolitan areas as in the rest of the state.

3. Continuity of supervision by health conferences, as measured by number of visits per patient, was well up to standard in metropolitan areas but much lower in the rest of the state.

4. Conference sessions in metropolitan areas were overcrowded.

5. In 31 of the 64 parishes of the state, containing 34 per cent of the state's newborn children, no child health conferences were available.

6. Conferences in metropolitan areas were held chiefly by pediatricians, in the rest of the state by general practitioners and health officers.

7. Public Health nursing services in the state amounted to 20 per cent of estimated need and about 60 per cent of the national average. Services in metropolitan areas, while better than the statewide average, were 20 per cent lower than the national average.

8. School health services were widely distributed, but 17 per cent of the colored children and 13 per cent of the white children in the state lived in parishes without any school medical service.

9. Dental clinic service was meager and available in only six parishes of the state.

10. Special clinics, such as those for mental hygiene, rheumatic fever, speech and hearing defects, were either nonexistent or not generally available.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

M. D. Hargrove, M. D., Ex Officio
C. Grenes Cole, M. D., Chairman
E. L. Leckert, M. D., Vice-Chairman
Sam Hobson, M. D., Secretary
C. M. Horton, M. D.
Leon J. Menville, M. D.

EDITORIAL STAFF

Philip H. Jones, M. D.....*Editor*

COLLABORATORS—COUNCILORS

Edwin L. Zander, M. D.
Joseph S. Kopfler, M. D.
Guy R. Jones, M. D.
Paul D. Abramson, M. D.
George Wright, M. D.
W. E. Barker, Jr., M. D.
C. A. Martin, M. D.
O. B. Owens, M. D.

Paul T. Talbot, M. D.....*General Manager*

1430 Tulane Avenue

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

VIRUS HEPATITIS

INFECTIOUS HEPATITIS

Infectious hepatitis is a term to include catarrhal jaundice, epidemic jaundice, and acute yellow atrophy of the liver. Circumstances were propitious for the study of various jaundiced states in the recent war. Many problems were solved. The above-mentioned conditions were shown to represent phases of a single disease entity. A new concept of the condition being due to a virus was evolved.

Haven (Medicine, September 1948) has presented a comprehensive and clarifying review of this subject. He discusses the forms of viral hepatitis: (1) Infectious hepatitis covering the concepts indicated above. (2) Homologous serum hepatitis, covering late postarsphenamine jaundice, syringe jaundice, transfusion jaundice, yellow fever vaccine jaundice. Whether or not the strains of virus producing the two types are different is not known. It is clear that the manifestations are sufficiently different to be separately considered.

It is seen that infectious hepatitis was recognized in the ancient world and has recurred sporadically and epidemically in civilian and military history since. It was a problem in Napoleon's troops in Egypt, in the Civil War, and in World War II in the troops stationed in the Mediterranean littoral. It occurs sporadically, endemically throughout the world. It is felt that the disease is more prevalent where sanitary conditions are poor. There is a tendency for an increased incidence in the autumn and early winter months.

Infectious hepatitis is primarily a disease of childhood in civilian life, but may occur at any age. Those under 15 are most susceptible. Among troops in World War II, those up to 30 were very susceptible, and after age 33 the incidence declined sharply.

The method of transmission is not known. Epidemiologic and experimental evidence suggests that some form of person-to-person contact is frequently operative. Various outbreaks have been considered as food-borne, milk-borne, and water-borne. Spread by the respiratory route has not been proved. The disease may be transmitted by the injection of as little as 0.01 cc. of infectious serum; accordingly, insect vectors have been considered, but not proved, as a means of transmission.

There is no susceptible laboratory animal and no specific serologic test. Much of the available information on virus hepatitis was obtained from experiments on volunteers. Convalescents from experimentally induced infectious hepatitis were immune when reinoculated with the homologous strain. Similar convalescents from infection with a Pennsylvania strain were im-

mune to the virus from a stool of a soldier who contracted jaundice in Italy.

The most striking effect of hepatic virus in man is in the parenchymal cells of the liver. It is a generalized infection. There are also lymphadenopathy, splenomegaly, inflammatory changes in the intestinal tract, and evidence of anemia. Biopsies in various stages in nonfatal cases revealed a number of inflammatory and degenerative changes in liver cells. Recovery takes place usually in one to two months, but may be much longer. Nodular hyperplasia, postnecrotic cirrhosis, and portal cirrhosis have been recorded in patients who have had infectious hepatitis. The frequency of this transition to cirrhosis is not known. In the fatal forms of the acute disease, the progression may be fulminant within ten days, or subacute over three to eight weeks It has been demonstrated that the etiologic agent is filterable, resistant to heating to 56° C. for thirty minutes, and transmissible to man by feeding or by parenteral inoculation of infectious material.

The clinical course of the disease is milder in children than in adults. The incubation period is probably twenty to forty days but may be as short as ten days. In the child, the condition appears abruptly with headache, chills, fever, abdominal pain, and vomiting; diarrhea may develop. The temperature ranges from 100 to 102° F., and after four to five days declines. At this time jaundice appears and persists ten to twelve days, followed usually by recovery.

In the adult there is usually a pre-icteric phase, as in the child, with the symptoms somewhat more intense. Anorexia is the most common and usually the earliest symptom. Jaundice may appear in the febrile period. The duration of jaundice is three to five weeks but may extend to four months. In the pre-icteric phase, the examiner may find some posterior cervical lymphadenopathy, moderate injection of conjunctivae, tenderness in the right upper quadrant, and enlargement of the liver. The liver frequently becomes tender. The mortality is less than 4 per 1000. Relapse may occur, and in such a case jaundice may or may not appear. Chronic hepatitis may persist in a small group and has been reported as occurring in 5 and in 2.3 per cent.

Laboratory studies have revealed that the erythrocytes are usually normal, but after debilitating illness, anemia may develop. There is a lymphopenia and neutropenia, early followed by a return to normal. The atypical lymphocyte can not be differentiated from similar cells considered diagnostic of infectious mononucleosis. Sedimentation index is not increased early in the disease but is increased on the third day of malaria. Certain observers report 20 per cent false positive Wassermann and Kahn reactions, and others, 1.5 to 2.5 per cent. Liver function tests show significant alterations. The bromsulfalein test may become positive as early as the third day. Roentgenographic studies in volunteers have shown evidence of gastroduodenitis. Gastroscopic examinations similarly have revealed small aphthous ulcers and acute gastritis. Transient electrocardiographic changes have been observed.

The differential diagnosis before the appearance of jaundice must cover the acute enteric fevers, malaria, dengue, infectious mononucleosis, and appendicitis. During the icteric phase, biliary sepsis, Weil's disease, yellow fever, malaria, and carcinoma of the pancreas, or other forms of biliary obstruction may be confused in the differential diagnosis. More remotely to be considered are the septicemias, amebic hepatitis, hemolytic jaundice, and jaundice resulting from chemical poisoning.

The treatment of this condition is symptomatic. Human gamma globulin has no value when given after the onset of the disease. Bed rest and adequate diet are the important provisions which must be obtained. During phases of anorexia and vomiting the usual supportive measures should be instituted. In convalescence, 3000 to 4000 calories are desirable.

The prognosis is usually good but less favorable in those patients who have a history of previous disease of the liver. Specific measures for community prevention

and control are not known. Adequate sanitation is of value here as elsewhere. Individuals may be protected if normal human gamma globulin is given six days before the onset of the disease. For the average adult, 10 cc. is sufficient.

HOMOLOGOUS SERUM HEPATITIS

Homologous serum hepatitis, as indicated in the preceding discussion, is a virus hepatitis, and the relation to infectious hepatitis is not clear. There were 51,337 cases in the U. S. Army epidemic of 1942, following the administration of icterogenic yellow fever vaccine. Numerous outbreaks have followed the use of other vaccines. The virus passes Berkefeld and Seitz filters and is resistant to temperatures of 56 to 60° C. for thirty minutes. It is parenterally transmissible to volunteers in serial passage and evokes homologous immunity. The incubation period varies from four to twenty-five weeks, and averages forty to one hundred and sixty days. The evidence is that parenteral transmission is the most important route. There is no evidence of cross immunity between homologous serum hepatitis and infectious hepatitis. The period of infectivity of patients with this disease is not known.

It is seen from these considerations that the existence of virus hepatitis presents a vastly different concept of jaundice due to a disease-producing organism and its pathology in the liver.

—o—

DIABETES WEEK
December 6-12, 1948

If every person would visit his physician for a routine physical examination each year, many diseases would be detected in the early phase, and treatment started at such a stage would bring results more satisfactory to patient and physician. However, because of the unwillingness of the public to utilize this method to its proper extent, many individuals continue to have undetected diseases. Consequently, special societies have resorted to the logical device of focussing the attention of the community on particular diseases, or groups of diseases, such as tuberculosis, cancer, heart disease, diabetes, and recently, multiple sclerosis.

To this end, the American Diabetes Association has proclaimed December 6-12, 1948, as "Diabetes Week."

In a recent survey of a Massachusetts city it was found that for every patient with diabetes, known and under treatment, there was another in whom the condition was unrecognized. To find who are the other 50 per cent of the community diabetics, the Association will sponsor the distribution of the necessary information, and will attempt to stimulate the public to have the needed examination.

Dr. A. A. Herold of Shreveport is organizing the program in Louisiana. This movement will require strong support from the physicians and will promote the best interests of the patient. Dr. Herold will need and appreciate the help of organized medicine.

—o—

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

1949 ANNUAL MEETING

Plans are now underway for development of the scientific program and other features of the 1949 Annual Meeting of the State Society which will be held at the Roosevelt Hotel in New Orleans on May 5-7. The Executive Committee has decided to hold the meeting next year on Thursday, Friday and Saturday instead of during the first part of the week and it is felt that there will be

many advantages to such an arrangement. The first meeting of the House of Delegates will be on Thursday, May 5 and the scientific program will follow, beginning on the night of May 5 and continuing through Saturday, May 7. There was recently held a meeting of the various scientific sectional chairmen at which time an outline of the proposed program was made. This includes an open meeting on Thursday night possibly with scientific papers presented by guest speakers and symposia on Friday morning and afternoon. Specific subjects have not been selected for these symposia, however it is probable that one of these will deal with traumatic surgery. On Saturday morning and afternoon there will be held meetings of the various specialty groups; some of the smaller groups meeting together, as suggested by the chairmen of these sections. Luncheons will be held by some of these specialties on Saturday in addition to the regularly scheduled luncheons for the House of Delegates and general membership of the State Society which tentatively are planned for Thursday and Friday respectively. Any member desiring information concerning a specific scientific section should contact the chairman of the section in which he is interested. Following is information concerning the doctors who have been appointed to serve in this capacity.

Dr. Emma S. Moss, New Orleans— Bacteriology and Pathology.

Dr. Kenneth Jones, Shreveport—Ear, Nose and Throat.

Dr. P. L. Perot, Monroe—Eye.

Dr. Murrel Kaplan, New Orleans—Gastro-enterology.

Dr. C. Gordon Johnson, New Orleans—Gynecology.

Dr. Edgar Hull, New Orleans—Medicine.

Dr. D. L. Kerlin, Shreveport—Neuropsychiatry.

Dr. C. Raymond Mays, Shreveport—Obstetrics.

Dr. C. R. Reed, Shreveport—Orthopedics.

Dr. W. C. Rivenbark, New Orleans—Pediatrics.

Dr. W. L. Treuting, New Orleans—Public Health.

Dr. G. M. Riley, Shreveport—Radiology.

Dr. J. E. Heard, Shreveport—Surgery.

Dr. C. O. Frederick, Lake Charles—Urology.

Dr. Robert F. Sharp, of New Orleans, has been appointed by the President of the Orleans Parish Medical Society to serve as General Chairman of the Committee on Arrangements and has already been active in initiating plans for the meeting. The chairmen of sub-committees have not yet been appointed. When data are received in this regard it will be carried in the Journal so that members may contact the proper persons when desiring information concerning various phases of the meeting.

Floor plans for technical exhibit space have not yet been completed, however companies which have previously exhibited at meetings of the State Society have been contacted and from all indications we can expect a full participation by these companies which add considerable interest as well as financial assistance to these meetings.

It is hoped that many members of the organization are making plans to attend this, the sixty-ninth meeting of the Louisiana State Medical Society.

———o———

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SECOND DISTRICT MEDICAL SOCIETY

The regular meeting of the Second District Medical Society was held Thursday night, October 21 at the Wigwam Village, 4800 Airline Highway, Dr. Joel Gray, president, presiding. After a short business session Dr. Joseph Reddoch of New Orleans presented a most interesting paper on "Face Presentation."

FOURTH DISTRICT MEDICAL SOCIETY

Following supper in the hospital dining room, the Fourth District Medical Society meeting was held in the Assembly Room of the Shreveport Charity Hospital, Tuesday evening, October 5.

Dr. Ashton Graybiel, Captain in the United States Navy and head of the Research Department, Pensacola, Florida, was the essayist. In addition to pointing out some of the experimental work in progress in the Research Department, he discussed neurorespiratory asthenia and his observations on this interesting condition.

The following officers were elected for the year:

Dr. W. C. Gray, Springhill, President

Dr. R. B. Van Horn, Mansfield, Vice-President

Dr. Joe E. Holoubek, Shreveport, Secretary-Treasurer

Dr. L. S. Huckabay, Coushatta, Delegate to the State Society

Dr. J. C. Sanders, Shreveport, Alternate Delegate.

POSTGRADUATE COURSE ON NEOPLASIA

A postgraduate course on neoplasia is to be given by Tulane University, division of Graduate Medicine, November 15-17, 1948. The schedule of papers extends over the three day period. All meetings will be held in the Hutchinson Memorial auditorium except those on the last afternoon, which will be in the Miles Amphitheatre at Charity Hospital.

Two guest speakers will discuss special phases of the cancer problem. Dr. Vernon David of Chicago will give a paper on "Etiological Factors in Carcinoma of the Large Bowel." Dr. John McDonald of the Mayo Clinic will discuss "The Smear Technique as an Aid in the Diagnosis of Malignancy." These two presentations will constitute the first Annual Lectureship of the Louisiana Cancer Society. Thirty-three other papers in the field of malignant disease will occupy the remainder of the time allotted. Nearly every phase of malignant disease as it concerns the clinician will be covered. The subject matter has been selected in such a way as to present the prospect of a thorough discussion of established principles, recent contributions, and controversial subjects of practical importance.

L.S.U. MEDICAL ALUMNI ASSOCIATION NEW ORLEANS DISTRICT

The New Orleans District of the L.S.U. Medical Alumni Association entertained its membership and their guests at the first of their annual dances on September 24 at the Jung Hotel. Over 400 persons attended; many of the graduates who are at present practicing in various South Louisiana communities were among them.

The guests of honor were the members of the present Senior Class of the School of Medicine, the Dean, and the Heads of the various Departments of the School. Also present were the Officers of the L.S.U. Metropolitan Alumni Association.

Before the dance several private cocktail parties were given by individual members which helped to make a complete evening of the affair.

It was announced that the next meeting of the New Orleans District would be held in the first part of January at a downtown center and that the State Medical Alumni Association had asked the New Orleans District to serve as host at a cocktail party to be given during the May meeting of the Louisiana State Medical Society. All graduates who attend the State Medical Society Meeting are invited to attend this party.

The committee for the dance included Dr. Jack R. Anderson, President of the New Orleans District, Dr. Elliot Roy, Vice-President, Dr. Philip Cenac, Secretary-Treasurer, Dr. Paul Getzoff, Dr. O. R. Depp, Dr. Marion LeDoux, Dr. Harry Brian, Dr. Lawrence Kavanagh, Dr. Robert Simmons, and Dr. Burchell Liles. Dr. Elliot Roy was in charge of arrangements.

SOUTHERN PSYCHIATRIC ASSOCIATION

The tenth annual meeting of the Southern Psychiatric Association will be held in Dallas at the Hotel Adolphus December 6-7. The President, Dr. Guy F. Witt, Dallas, Secretary-Treasurer, Dr. Newdigate M. Owensby, Atlanta, and the Chairman of the Committee on Arrangements, Dr. Arthur J. Schwenkenberg, Dallas, extend a cordial invitation to psychiatrists of Louisiana, including those connected with the Veterans Administration and the U. S. Army, to be present. Although the Association is designated as a psychiatric association it is made up of both psychiatrists and neurologists and several papers will be given in neurology and other allied fields.

WOMAN'S AUXILIARY

The Woman's Auxiliary is indeed grateful to the editors of the New Orleans Medical and Surgical Journal for the roster of its members with addresses printed in the October issue. All members should clip this entire section for future reference. It serves as a substitute for a Year Book which your Year Book Chairman, Mrs. S. L. Calhoun, has so capably compiled for you.

The October issue of "News and Views" has been mailed. If you have not received your copy please advise Mr. Frank Lais, Jr., 1430 Tulane Ave., New Orleans of the omission, with your name and address.

Year Books have been received by your chairman from the Woman's Auxiliary to the East Baton Rouge Parish Medical Society and to the Rapides Parish Medical Society, Alexandria. Programs for both auxiliaries have been planned for the year to include in some way all of the state projects for the year 1948-1949, namely: 1. Assisting Council on Medical Service; 2. Education—Membership, Laity; 3. Legislation; 4. Rural Medical Service; 5. Medical Cultural Items; 6. Hygeia; 7. Organization; 8. Health Days; 9. Red Cross; 10. Cancer; 11. Doctors' Day. Clippings from both auxiliaries reported interesting programs for the October meetings. The Alexandria Auxiliary based its program on articles from Hygeia and the Baton Rouge Auxiliary heard from the President of its Medical Society, Dr. John T. Lewis, who outlined a splendid Public Relations program.

A letter from the auxiliary to the Iberville Parish Medical Society, Plaquemine, stated plans to sponsor a boy and girl for Pelican State in 1949. The Orleans Parish Auxiliary maintains a history book with news and pictures of the auxiliary throughout the year instead of a year book. Cards are to be filed stating whether members have their annual check-up or not. A drive among members for both Tuberculosis and Community Chest through auxiliary chairmen is now taking place.

MRS. F. U. DARBY,
Press & Publicity Chairman

BOOK REVIEWS

Practice of Allergy: By Warren T. Vaughan, M. D. Revised by J. Harvey Black, M. D. 2d ed. St. Louis, C. V. Mosby Co., 1948. Pp. 1132. Price, $15.00.

Warren T. Vaughan, son of Victor Vaughan of Ann Arbor, became interested in clinical allergy shortly after World War I. At that time the specialty of allergy was in its infancy. The pioneer work of Noon and Freeman of London in 1911, had just shown that people with grass pollen hay fever could be benefited by specific hyposensitization. Allergy was a frontier of medicine and men like Vaughan, Cooke, Black, and Coca were drawn to it. With the passage of the years Vaughan made many important contributions. His clinic in Richmond was well staffed and equipped.

In 1930 he published the first edition of "Allergy and Applied Immunology". This was followed by a second edition in 1934. His later works "Strange Malady" and "Primer of Allergy" were intended primarily for the instruction of his patients. There was a second edition of the "Primer".

The first edition of "Practice of Allergy" appeared in 1939. This book at that time was the most comprehensive publication on allergy ever printed, and was a veritable storehouse of clinical observations. Many of the excellent illustrations were made by Vaughan himself with his Leica. The personal opinions of the author enriched the material. The reviewer read the book from cover to cover and has never regretted it. Many allergists spoke of it as their "Bible". The book had a wide demand, and later went out of print and became unobtainable.

Before his untimely death in 1944, Dr. Vaughan was collecting material for the second edition.

This task was later passed on to his friend and contemporary, Dr. J. Harvey Black of Dallas, who is himself one of the pioneers in allergy. He started in pathology, and later became interested in the budding specialty of allergy. He also has made many eminent contributions to the literature. He is highly regarded by all who know him, he is extremely modest, and many honors have rightfully been bestowed on him. He has always been sympathetic toward the problems of the younger men in allergy. He has held positions of honor in various national and sectional allergy societies.

The second edition of Vaughan's "Practice of Allergy" greatly resembles the first edition. The same illustrations are there. Much of the written material is unchanged. Black in his preface states that, "We have, in every instance, tried to retain the quality and the flavor of the book so that it might remain as it was written, 'Warren Vaughan's book.' It was the quality and the flavor which he gave it that made for its great popularity, and we hope it remains as distinctive as it was when he wrote it. We have added new material which has appeared, and removed any which, by the passing of years and accumulation of knowledge, has been made unacceptable. Where there is room for honest difference of opinion and his opinion differed from ours, we have retained his. Where advancement in our knowledge has made an opinion no longer tenable, we have not hesitated to change it as he would have changed it if he had lived to see this day."

In the book we have therefore the advantage of Vaughan's ideas and well organized material, which has been changed when needed to fit in with any altered viewpoints. New contributors appear in the second edition. O. C. Durham wrote the chapters on Field Surveys and Aerobiology. J. B. Howell rewrote the chapter on Fungus Infection With Associated Allergy. James Holman rewrote the chapter on Vital Capacity.

Your reviewer wonders why no mention was made of the study of Petersen and Vaughan on the connection of weather changes and fatal cases of asthma. This report appeared shortly before Vaughan's death, and it was not discussed in the second edition. Possibly Dr. Black has certain opinions on this topic which do not agree with those of the article.

Your reviewer feels that this new work will serve as a most valuable reference. The young man interested in clinical allergy will do well to read this book. He should absorb the opinions and viewpoints of the two authors. The dynamic personality of Warren Vaughan still lives.

HENRY OGDEN, M. D.

Motor Disorders in Nervous Diseases : By Ernst Herz, M. D. and Tracy J. Putnam, M. D. New York, King's Crown Press, 1946. Pp 184. Illus. Price, $3.00.

Occasionally a medical publication appears which is so superior to other works of its kind that it is destined to be looked upon as authoritative for many years to come. Dr. Ernst Herz and Dr. Tracy Putnam of the Department of Neurology, College of Physicians and Surgeons of Columbia University, have succeeded admirably in presenting a concise yet complete discussion of abnormalities of the motor system.

All phases of the subject are discussed. Gait, involuntary movements, incoordination, reflexes and motor disorders of the cranial nerves are thoroughly covered. Clinical pictures are vividly presented by the use of strips of motion picture film throughout the text. Photographs, diagrams and anatomical drawings are used to illustrate the various problems of discussion.

One might wish that the authors had used more illustrations of neuropathological preparations to supplement the various case presentations. Beyond this no further criticism can be offered. To all who are interested in any phase of neurology this work is unhesitatingly recommended. To the reviewer it seems destined to become a classic.

JOHN W. BICK, JR., M. D.

Fatigue and Impairment in Man: By S. Howard Bartley and Eloise Chute. New York, McGraw-Hill Book Company, Inc., 1947. Pp. 429. Price, $5.50.

As the authors state in their introduction, "Fatigue as an unpleasant experience has entered into the life of everyone." In spite of a considerable amount of work on the subject, however, very little definitive evidence is available as to its causation and characteristics. In this volume, the authors have brought together a considerable amount of physiological and psychological work and have attempted to evalute its relationship to the problem.

It is pointed out that even a definition of fatigue is difficult. The common concept that fatigue is synonymous with impairment is shown to be unsound. The various definitions that have been used in physiological and psychological literature are examined in an attempt to arrive at an adequate definition. Considerable data is given on the various factors which have been shown to limit activity, such as anoxia, lack of sugar and salts, and temperature. Metabolism and nutrition are discussed and there is a splendid chapter on visual-fatigue studies. Hours and conditions of work in industry are discussed briefly. Of particular interest is a review of drug action in relation to fatigue or impairment. The common drugs such as caffein, benzedrine, and alcohol, are considered and the

authors emphasize the scarcity of data as to the actual effects of these drugs on the individual insofar as fatigue is concerned. Sleep is discussed at length as are the various factors which influence it. The authors conclude that the primary function of sleep is to provide for various sorts of realignment that cannot occur while the organism is in the midst of high activities.

The latter part of the book is concerned with a review of psychological factors concerned in fatigue, such as training and conditioning, aging, "mental fatigue", personal factors in the situation, visual performance and fatigue, conflict and frustration, and chronic fatigue. In their conclusion, the authors point out that their monograph has served to indicate the necessity for considerably more work on the complex problem of fatigue. They reiterate their plea that future work on fatigue should be concerned with the experience of feeling tired rather than on specific limiting factors which so far have been important in showing what fatigue is not. The authors feel that the subjective phenomona generally have been given little consideration and yet are extremely important in the evaluation of the condition.

This is an unusually good monograph which will be of value to physiologist, psychologist and clinician. The latter will find it extremely valuable in providing him with a good review of the literature and, perhaps more important, in giving him a sound point of view on a subject in which he should be interested and well informed.

H. S. MAYERSON, PH. D.

A Biology of Disease: By Eli Moschowitz, M. D. New York, Grune & Stratton, 1948. Pp. 221. Price, $4.50.

In the words of the author, the main purpose of these essays "is to stimulate a point of view and a methodology rather than to be strictly informative, and to emphasize the dynamic as opposed to the static approach in the study of disease." He considers that in the absence of a "knowledge of etiology, one of the best bases for the classification of chronic disease is a uniform and consistent pathogenesis." He makes an effort to show that diseases pursue a long course, and that it is frequent for investigators to pick out phases in the course of the disease and treat them as distinct syndromes rather than part of the whole. The contents of the book deal with twenty four medical topics. They are all ably presented and reveal the author to be authoritative in his chosen field. One cannot but feel, as he reviews the book, that here is a most timely subject, especially in an era of medicine when, in the words of Sir Thomas Lewis, "the continued separation of disease into types can be pressed too far . . . and thus hinder progress in studying disease."

I. L. ROBBINS, M. D.

A Manual of Clinical Therapeutics: By Windsor C. Cutting, M. D. Philadelphia, W. B. Saunders, 1948. Pp. 712. Price, $5.00.

This is a second edition quite satisfactorily brought up to date. The author presents "the workable facts of therapeutics as briefly and succintly as possible." Since it is intended as a guide for students and practitioners it covers a broad variety of subjects. It should be quite helpful and merits the attention of one interested in this type of book.

I. L. ROBBINS, M. D.

Skeletal Tuberculosis: By Vicente Sanchis-Olmos, M. D., (translated from the Spanish by John G. Kuhns, M. D.). Baltimore, The William and Wilkins Company, 1948. Price, $5.00.

This book presents in the English language many European doctrinal concepts and scientific judgments. It contrasts the methods of treatment and clinical concepts of two continents. The purpose of the publication is stated to be "with the intention of emphasizing that which is new or little known."

The book is divided into two parts, the first dealing with pathogenesis, pathology, etiology, and basic considerations in diagnosis, prognosis and treatment. The second part, entitled "Special Subjects", considers the various bone and joint structures individually.

In the first part, the author's vast working knowledge of the European and American medical literature is evident, although it is frequently presented in such abbreviated form that the reader gains the impression that the material was intended primarily for the expert who is already conversant with the European concepts. Much of the discussion is theoretical, but it at least gives the average American reader some insight into the European surgeons' line of reasoning.

The second part, dealing with the various anatomical areas, suffers from constant repetition of the pathology, roentgenographic appearance, symptomatology, differential diagnosis and prognosis. Much of this material could be condensed without detracting from the value of the book as a surgical reference. There is no mention of the use of streptomycin as an aid in the management.

The book is of little practical value to the practicing surgeon but should be of great value as a stimulating reference work for the real student of skeletal tuberculosis.

R. M. KIMBALL, M. D.

Foundations of Neuropsychiatry: By Stanley Cobb, M. D. 4th ed. Baltimore, William and Wilkins Co., 1948. Pp. 260. Price, $2.50.

This is a fourth revised and enlarged edition of

the work formerly known as "A Preface to Nervous Disease." In it, psychiatry is conceived of as a pyramid, with the basic sciences as a foundation, and philosophy as the apex. As the author points out, the mental sciences are as yet only feebly supported by the fundamental sciences. Nevertheless, a great deal of material has been gathered by observation; but, because this book is designed for use as a preface to further studies in neurology and psychiatry, the author has limited his discussion to only those principles that are fairly well established, and in all cases has correlated the anatomic, physiologic, and pathologic data. There are thirteen chapters, on the autonomic nervous system, the cerebrospinal nervous system, motor integration and locomotion, the cerebral cortex, consciousness and the "mind-body" problem, cerebral circulation, cerebrospinal fluid, general neuropathology, the peripheral nerve and neuritis, special neuropathology, epilepsy, some psychological concepts important in medicine, and psychopathology. Although the small size of this volume causes this material to be served up in very concentrated form, it can, in general, be assimilated without difficulty by the student. The bibliography is well-chosen. The book should be of value to both the student and the practitioner, as it attempts, with some success, to give a broad and integrated view of the anatomy, physiology, and pathology of the brain. Because the problem of neuropsychiatry is such a great one, any volume, such as this, which will stimulate interest in the field, is a welcome one.

T. TREUTING, M. D.

Neuroanatomy: By Fred A. Mettler. 2nd ed. St. Louis, The C. V. Mosby Co., 1948. Pp. 536. Price, $10.00.

This is an excellent text and reference work, especially for advanced students in neurology. It is superbly illustrated in both the gross and microscopic portions. The expanded sections on vascular supply are noteworthy additions. The text has also been brought up to date by the inclusion of the results of recent world-wide neurological investigations.

The general plan of presentation is the same as that used in the first edition. Part one is devoted primarily to the description of the gross anatomy of the spinal cord and brain. Part two contains the minute description of microscopic sections of ascending levels of the neuraxis, cerebellum and cerebral hemispheres. Functional considerations are taken up as the nuclei and tracts are passed by in the various levels.

To those who believe this approach to the subject is desirable, this book should prove to be of great value. However, teachers of short introductory courses given to first year medical students will probably find the text somewhat unwieldly, especially if they prefer to emphasize functional systems with anatomical details cut to a minimum.

THEODORE SNOOK, PH. D.

PUBLICATIONS RECEIVED

The American Cancer Society, New York: Cancer Current Literature (Vol. 2, No. 3, Mar. 1948), prepared by the Library of The Medical and Scientific Department.

American Nurses' Association, Inc., New York: ANA Public Relations Workshop.

Appleton-Century-Crofts, Inc., New York: Abdominal Operations (Second Edition) by Rodney Maingot, F. R. C. S. Eng.

Doubleday & Co., New York: Your Baby, by Gladys Denny Shultz and Lee Forrest Hill, M. D.

C. V. Mosby Company, St. Louis: British Surgical Practice (Volume 3), by Sir Ernest Rock Carling, F. R. C. S., F. R. C. P. and J. Paterson Ross, M. S., F. R. C. S.

Oxford University Press, New York: Detailed Atlas of the Head and Neck, by Raymond C. Truex, M. S., Ph. D. and Carl E. Kellner.

W. B. Saunders Company, Philadelphia: A.M.A. Interns' Manual. Psychiatry in General Practice, Melvin W. Thorner, M. D., D. Sc.

South Carolina Medical Association, Charleston: A Brief History of the South Carolina Medical Association, by Joseph Ioor Waring, M. D. An Account of the Weather and Diseases of South Carolina, by Lionel Chalmers, M. D.

The Williams & Wilkins Company, Baltimore: A Method of Anatomy (Fourth Edition), by J. C. Boileau Grant, M. C., M. B., Ch. B., F. R. C. S. (Edin.). Occupational Therapy Source Book, edited by Sidney Licht, M. D. Elementary Anesthesia, by W. N. Kemp, M. D. C. M.